THE MEDICAL JOURNAL OF AUSTRALIA

VOL. I.—28TH YEAR. SYDNEY, SATURDAY, MARCH 29, 1941. No. 13.

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WARFARE AND TROPICAL MEDICINE.[1]

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SIR PATRICK MANSON, who was a fine physician if a poor epistemologist, entitled his *magnum opus* "Tropical Diseases". Those of you (and no doubt in this enlightened city it is the majority) who are pioneers in the Neo-Hippocratic revival—who, following the strictures of Ogden and Richards, have put aside for ever all loose talk about "resistances", "immunities" and "toxæmias"—will be well aware that there are no such things as "diseases", much less "tropical diseases"; and that that of which I am about to talk to you are those syndromes which for one reason or another tend to be more frequently encountered between arbitrary lines known as Cancer and Capricorn. It will be my endeavour to discuss why these certain syndromes are more common, and what procedures, based on the factors concerned, may be expected to prevent them from involving groups of men brought temporarily into these areas for the purpose of conducting warfare.

For some reason, not particularly clear, persons living in temperate climates imagine that the tropics are very hot. Such is, of course, not necessarily the case. Summer temperatures in Perth are far higher than in Batavia. The characteristic of the tropics is that the length of the day varies but slightly or not at all throughout the year. Otherwise climatic conditions depend on factors not concerned with degrees of latitude—on land mass, soil, prevailing winds and rains, altitudes—and on secondary factors such as population, flora, fauna; and it is to these factors that we must look in considering disease in relation to these areas.

There is good reason to believe that gregarious life among men, of a kind that has come to be associated with the word "civilization", was first developed about the upper reaches of the Indus River, and from this point spread east and west, until along a line from Cairo to Pekin had arisen a series of organizations which even today, after some 5,000 years, still comprise the most important part of the human race. This part accounts for more than 1,000,000,000 beings and is now increasing at a rate of perhaps 20,000,000 a year, with no signs of diminishing vitality such as is obvious in the materially more spectacular, if fundamentally less perfectly integrated, far western extension of the same process, of which we present here tonight are products.

It so happens that a great part of the Indian and Chinese civilizations, together with their immediate extensions, lie within or near to the tropical belt, and it thus came about that when western medicine was developed in these areas and concerned itself with the special problems arising among these peoples, this aspect of it came to be loosely known as "tropical medicine" and is so known today, though added to it has been the study of disease as seen among the more primitive peoples of Central Africa and Central America.

If we study mortality statistics among the peoples of the older civilizations I have mentioned, we find a number of clearly defined manifestations of disease that account for the greater part of their death rate: pneumonia, malaria, cholera, plague, physical injury, starvation, tuberculosis, bacillary dysentery, syphilis, typhus, smallpox, kala-azar, ankylostomiasis.

Central Africa and Central America have yellow fever and trypanosomiasis as problems of their own, and there are other conditions less common, less fatal, or less widely distributed that must be taken into account in this discussion; amœbiasis, enteric fever, leptospirosis, filariasis, schistosomiasis, leprosy, gonorrhœa, brucellosis and relapsing fever are among them.

[1] Read at a meeting of the Western Australian Branch of the British Medical Association on September 18, 1940.

Now these conditions fall naturally into certain groups. Starvation and physical injury are an integral part of competition for survival. Syphilis, gonorrhœa, and the three other venereal infections are associated with defects of social organization, perhaps of less importance than those at present bringing western civilization towards extinction. The dysenteries, enteric fever, cholera, leptospirosis and some helminth infestations are associated with the ingestion of the fæces or urine of human beings or animals. Pneumonia, tuberculosis, pneumonic plague, smallpox, brucellosis, leprosy and possibly kala-azar are the outcome of the inhalation or swallowing of salivary spray, or of exudates or secretions from dermal lesions. Malaria, bubonic plague, yellow fever, trypanosomiasis, filariasis, typhus, relapsing fever and probably kala-azar are transmitted by insects. In the case of ankylostomiasis, schistosomiasis and some other helminth infestations, the parasites concerned are capable of penetrating the skin of their future hosts by their own endeavours.

The important forms of disease in western civilization constitute a very different picture. Conditions of housing and hygiene are such that today these major infections are becoming steadily rarer, and men do not often succumb to such directly or simply transmitted agents. They succumb rather to degenerations associated with physical, mental and moral deterioration, such as the psychoses, the psychoneuroses, cancer, osteoarthritis, arterial sclerosis and such allied conditions as hypertension, coronary thrombosis, chronic interstitial nephritis, diabetes, prostatic hypertrophy—conditions the prevention of and treatment of which are unlikely to be satisfactorily achieved through our present approach by the analytical methods in vogue in the sciences of physiology and pathology. But that is another story.

As the present war extends—if it does extend—to the Middle or Far East, masses of men may pass from the second disease environment that I have described to the first, and with their passing create special problems for those medical officers who may accompany them.

First, it must be emphasized that in passing from their own environment to another not their own, such men carry with them, since their own environment is characteristically that of intrinsic disease, all the liabilities that have already been incurred, except that by selection the grossly affected have been eliminated. Any one of them may develop insanity, diabetes or cancer; but in this very different milieu the physician may well find it difficult not to overlook this simple fact; in the face of forms of disease that are new to him, he may misinterpret those that in actuality are old. Tropical medicine may be described as western medicine to which the rest of medicine has been added.

Ideally, perhaps, an army transferred to a new setting might be kept isolated from the people in that setting and even from animal reservoir hosts. In practice this is not possible, and constantly the risk is present that disease endemic among the native population will spread as an epidemic among such men. They enter towns and villages and mingle in crowds, acquiring spray infections and vermin—they eat in restaurants—they consort with prostitutes—they bathe where natives have bathed, defæcated and urinated—and sooner or later disease is encountered. No large military force has yet entered the East, so far as I know, without suffering more casualties from disease than from wounds. From the Gallipoli campaign more than 80,000 sick men were withdrawn.

Let us consider, by way of example, some associations of the two symptoms, diarrhœa and dysentery; let us confine ourselves to associations that can be transmitted from man to man by the simple process of coprophagia. The agents of the so-called food poisonings are *Salmonella enteritidis*, *Salmonella aertrycke*, *Salmonella choleræsuis*; those of the dysenteries proper are *Shigella dysenteriæ* (Shiga-Krause), *Shigella ambigua* (Schmidt), *Shigella paradysenteriæ* (Flexner), *Shigella alkalescens*, *Shigella sonnei*; those of the enteric fevers are *Eberthella typhi*, *Salmonella paratyphosa* (*A* and *B*); that of cholera is *Vibrion choleræ*; that of amœbiasis is *Entamœba histolytica*. This is a considerable list of agents, all capable of decimating groups of men in unfavourable circumstances.

Consider their sources: patients with mild infections who have failed to take medical advice; patients with severe lesions, receiving medical treatment, from whom others through carelessness become involved; carriers who have recovered from symptoms, but still harbour the agent; carriers who never have had symptoms; carriers with mild but recurring symptoms who may be a danger for years on end. The fæces of all these are a menace; they may come to contaminate food, water or fingers in devious ways; flies may feed on them, then vomit or defæcate during their next meal, or their feet may transmit the agent passively.

Here are many possibilities; and though, when we are aware of them, much may be done to minimize their chances of operation, complete success is hardly to be expected, so that sooner or later one or more men are involved and the new problems of diagnosis and treatment arise.

In times of war, even at base hospitals, the laboratory diagnosis of disease that may become epidemic is fraught with technical difficulties, and this is particularly the case when dysentery and diarrhœa are concerned. Nevertheless, laboratory diagnosis is essential for the prevention of spread, and the correct treatment of patients can rest on no other foundation. The clinician can no doubt recognize cholera, bacillary dysentery or enteric fever when one or another takes its classical form; but too often they do not do so, and too often such classical forms are mimicked in the activity of other pathogens. Subtertian malaria is notorious in this respect and may give rise to symptoms that may imitate closely any one of them—the dehydration characteristic of cholera, the frequent passage of blood and mucus with abdominal pain and tenesmus typical of severe dysentery, the great mental and physical prostration of enteric fever. Unless the vibrion is seen or grown on culture from the fæces, the eberthella grown from the blood, fæces or urine, or its activity detected serologically, the shigella identified, or the entamœba, either in mobile or in vegetative state, seen or grown on culture, these important associates of disastrous epidemics will certainly pass undetected until the problems created by their activity have been many times increased by spread. Individual lives also will be unnecessarily lost, for few procedures can be more damaging to a patient suffering from Shiga dysentery than a course of emetine, while the preparation and early intravenous administration of hypertonic saline solution, with potassium permanganate by mouth, will save an important percentage of patients suffering from cholera. But to discuss in detail problems arising in the diagnosis and treatment of this group is in itself material for a volume. Since the last war great advances have been made, particularly in our knowledge of the antigenic structure of the enteric group of bacteria. Much of this work has important practical applications.

Let us turn to another problem—fever of sudden onset in one or more men, perhaps far from a base hospital, somewhere in Africa. There are a few premonitory symptoms—headache, nausea, loss of appetite—then a sudden rise of temperature, a flushed face, aching limbs, congested eyes, a confused mind. Possibilities will occur to the physician: malaria, epidemic or scrub typhus, influenza, dengue or sand-fly fever, yellow fever, leptospirosis, relapsing fever—a formidable list of conditions of widely varying import; something very different from the leisurely consideration of lymphatic leuchæmia against lymphosarcoma, while the case is debated over cups of tea. Perhaps there are no books to turn to—only the officer's general knowledge of the background and characteristics of these infections. Should a microscope, a slide and some Leishman stain be at hand, thick films may exclude malaria. The absence of a history of wading in swampy country or of bathing in stagnant water may make leptospirosis seem unlikely; yellow fever is generally confined to certain areas, and information may have been acquired that none has been reported. Away from towns, when the troops are marching in bush country, scrub typhus or relapsing fever would appear to be the most

probable alternatives; but *Stegomyia fasciata* may abound and dengue fever cannot be excluded. Two days later the patient's conjunctivæ are yellow and the fever is still high. Leptospirosis and yellow fever come back to mind; but relapsing fever associated with the African strain of the spirochæte (*Spirochæte duttoni*) is commonly accompanied by jaundice. Shall 0·8 gramme of "Novarsenobillon" be given as a speculation, dark ground illumination not being available? Or has this cycle gone too far, making the risk of a fatal reaction to the wholesale destruction of spirochætes too great, even if the diagnosis is correct?

Such is the type of problem that must constantly recur in the field during warfare in the tropics. Even at headquarters, where the aid of a pathologist may be available, the identification of the acute fevers presents difficulties. The virus infections—influenza, dengue fever, pappataci fever and yellow fever—are not yet commonly subject to laboratory diagnosis, though the rhesus monkey is susceptible to yellow fever, and blood from a patient may infect or passively immunize this animal, according to the stage of infection. *Spirochæta recurrentis* may be seen in the blood by dark-ground illumination during the stage of rising fever. On the other hand, *Leptospira icterohæmorrhagiæ* cannot be detected by this means, though in the first five days of illness blood injected into the peritoneum of a guinea-pig transmits the infection, and after the tenth day the patient's urine has the same effect. But even in the early stage this method of diagnosis is of limited value, since the guinea-pig takes several days to sicken; nor is diagnosis of great value to the patient, drugs such as arsenic having no effect. It may, however, be the means of tracing a source of the infection and of preventing other men from being involved.

In typhus the Weil-Felix test and, to a much lesser extent, the Neill-Mooser test, are of value. During wars it is louse-borne typhus that is feared; but during campaigns in open country, where rodents are prevalent, the tick-borne or mite-borne forms may be encountered, and in rat-infested towns the less fatal flea-borne type may become sporadic. Unfortunately, the agglutination of Proteus X19, X2, or XK by the serum of the patient cannot be expected to reach diagnostic titres for a week to ten days. This disadvantage apart, however, the reaction is of great significance.

I have already mentioned that offensive rodent, the rat, in connexion with three serious infections that may involve men on active service—scrub typhus, relapsing fever and leptospirosis. Louse-borne typhus, of course, does not necessarily require the presence of rodents, for an epidemic may originate from one man coming into contact with a lousy and infected native, and once initiated this form may be expected to spread with great rapidity unless "delousing" is most efficient.

There is, however, a disease of rats that is far more to be dreaded by armies, and that is plague. During times of peace in western countries one is inclined to think of plague as an infection of some historic interest—to think of the Black Death, and of the Great Plague of London that Daniel Defoe so vividly described. Unfortunately the activities of *Pasteurella pestis* are far from being confined to history, and in many parts of the East plague is still endemic and still prone to spread in epidemic form when conditions are suitable. Just as, when the strength of an individual is reduced by external factors, then some latent infection may spread through the tissues, so when society in the Old World is disintegrated by warfare or famine, then plague may spread and destroy that society, eliminating within a few months one-half to three-quarters of its members in an affected area.

It was for a long time believed that the common rat, *Rattus rattus* and *Rattus norvegicus*, were the two animal hosts responsible for all outbreaks of plague. Now it is certain that most rodents are liable to epizootics, just as different strains of pasteurella may cause violent epidemics in cattle, birds and other animals. In scrub country tarabagans (*Arctomys bobac*), small marmots (*Citellus citellus*), jerboas (*Dipodipus sagita*), gerbilles (*Rhombomys opimus*), ground squirrels (*Geosciurus capensis*) and multimammate mice (*Mastomys coucha*) have all been convicted of this activity; so that to regard plague as altogether a danger confined to towns is not correct, though the fleas abandoning dead rats are in practice the most likely to make the link between animal and man.

During times of war stores accumulate, often under unsatisfactory conditions, while the control of rats tends to become less and less efficient. An epizootic among them may pass undetected until the first human cases have occurred, and by then this epizootic may have reached such dimensions that its suppression may not be possible. Furthermore, among men debilitated by fatigue and malnutrition, those who contract severe bubonic infections may be so overwhelmed that they die of a pasteurella pneumonia; this form is directly transmissible by spray infection from man to man, and is unique among such infections in that recovery is so rare as to be of no practical importance. Though of recent years vaccines, serum and the sulphonamide derivatives may have altered the outlook in plague epidemics, it is difficult to believe that, should such epidemics occur in the present war, they will not be accompanied by serious loss of life.

Plague, typhus fever and yellow fever are notorious for their activity among physicians and other medical attendants. Hundreds of them have died from these infections in past epidemics, and should they fail to recognize early cases in the future this state of affairs will surely persist. If charity be held to begin at home, then those concerned do well to study these conditions in all their aspects.

Insect-borne diseases are not of great import in Australia—certainly not in those parts of Australia that carry the great majority of the population. In tropical countries and among the crowded communities of the Old World, however, the record of their activities is formidable.

Pediculus corporis vel capitis transmits epidemic typhus and the cosmopolitan type of relapsing fever that is associated with *Spirochæta recurrentis vel obermeieri*. Species of ticks—*Omithodorus moubata et lahorensis et tholozani* and *Argas persicus* transmit other strains of the relapsing fever spirochæte—*Spirochæta duttoni et persica et hispanica*. In the typhus group the tick *Dermacentor venustus et variabilis* conveys Rocky Mountain spotted fever, and *Rhipicephalus sanguineus* conveys the *fièvre boutonneuse* of the Mediterranean littoral. In the same area species of dog ticks transmit ponos, the Mediterranean type of visceral leishmaniasis. For mite-borne typhus of the tsutsugamushi and scrub types, *Trombicula deliensis et schuffneri et akamushi* among others are responsible. Fleas, more particularly *Xenopsylla cheopis*, *Ceratophyllus fasciatus* and *Ctenocephalus canis* transmit plague. These and other species also may convey typhus and less certainly ponos. Sandflies, more particularly *Phlebotomus argentipes et papatacii*, transmit sandfly fever and dermal and visceral leishmaniasis, though the latter when epidemic, like plague, possibly becomes subject to direct transmission by respiratory spray. Among mosquitoes *Stegomyia fasciata* conveys dengue and yellow fever; *Culex fatigans* carries *Filaria bancrofti*, while some fifty species of anopheles are known to transmit malaria. The mangrove fly, *Chrysops dimidiata*, conveys loaisis; the junga fly, *Simulium damnosum*, onchocerciasis; the tsetse flies, *Glossina palpalis et morsitans*, the two types of trypanosomiasis associated with *Trypanosoma gambiense* and *Trypanosoma rhodesiense*.

This is a formidable list of vectors, and to acquire a sound knowledge of the identification, distribution, habits and means of evading or destroying these insects is a formidable task. Yet to proceed to areas such as Central Africa with large bodies of men, but without such knowledge, is to court failure in their control.

Consider malaria alone as an insect-borne disease. In times of peace it kills annually the equivalent of four times the population of Australia, and incapacitates to a greater or lesser degree ten times that number. During wars, as social disorganization increases, these figures may be expected to rise steadily, and large bodies of men, all highly susceptible, may well be decimated unless active measures are taken to break the complex life cycle of the plasmodium. These problems alone might be the subject

of a dozen lectures, so many factors are involved. First there is the determination of whether an area is malarious or not. Information may already be available, or a thick film survey or a spleen index may be required. When a site is found to be both malarious and unavoidable, it is necessary to take into consideration the period for which it will be occupied and the intensity of infection likely. Thus, for a large camp to be maintained over months, the species of vector, its life habits and the infection rate present should be known, so that the site of the camp in relation to swamps, jungle, cultivated land and villages can then be decided. If on the other hand a small force is passing fairly rapidly through malarious country, "Atebrin" prophylaxis may be indicated. Other temporary expedients include the rendering of the skin unattractive to mosquitoes, or the spraying of quarters with a repellent. Individuals may also be protected mechanically from being bitten or be protected in groups. A campaign against adult mosquitoes with swats may often help to keep down infection rates.

When more permanent camps are concerned, it may be economic to drain breeding places by open channels, by subsoil pipes, by the burying of tree trunks (fascine drainage), by the filling of swamps or by the planting of vegetation that takes up water rapidly, such as banana palms or *Eucalyptus globus*.

Drainage may be too expensive, or the materials or labour may not be available. In such case larvæ may be destroyed by oil, whether sprayed, drip fed or diffused from oil-soaked materials. Alternatively, when the destruction of fish does not matter, the water may be poisoned with cresol; where this cannot be done the water may be sprayed with copper aceto-arsenate (Paris green). Finally, inflammable oils may be used and marshes burnt off with flame guns to destroy both adult mosquitoes and larvæ.

In practice, of course, it is usually expedient to combine several of these procedures in a campaign against malaria, and it must be clear that considerable judgement is involved in deciding which shall and shall not be emphasized in relation both to efficiency and cost.

This is just one problem in insect control. Lice, ticks, mites and the biting and non-biting flies may all demand control and prove a heavy tax on the ingenuity of medical officers, limited, often severely, by lack of materials and time, in the methods they can adopt.

Such are some examples of the epidemic diseases that may bring disaster to an army, whether spray-borne, fæces-borne or insect-borne. When man power is all important, the key position of the medical officer stands out clearly, and this has been well realized and acted upon by those in command of the Italian forces.

Over and above the epidemic infections, there are individual complaints, often of no very serious portent, that may sap the vitality and morale of troops. Heavy work in the heat may give rise to conditions such as siriasis (heat hyperpyrexia, a serious and often fatal condition), heat exhaustion (a type of shock), heat cramps (associated with salt deficiency), or sun traumatism associated with excessive exposure to actinic rays. The occurrence of such lesions necessarily lowers the standard of work among the other men and every precaution must be taken to prevent them from occurring.

Excessive heat or humidity is a severe test of the skin, and miliaria and epidermophytosis may become general. The latter is highly infectious and may spread in epidemic form. The medical officer in such areas must be well informed in the prevention, diagnosis and treatment of these conditions, for in the field during open warfare a severe outbreak of pedal tinea may immobilize part of a force.

Helminths are another and complex problem, and it is by no means easy to keep in mind the life histories and habits of all that may be concerned. Some pass directly from man to man—*Enterobius vermicularis, Trichiuris trichiura, Ancylostomum duodenale, Necator americanus, Ascaris lumbricoides*; some pass from animals to man or from man to man through an animal in the course of their life cycle—*Echinococcus granulosus, Trichinella spiralis, Dipylidium caninum, Fasciola hepatica, Hymenolepis nana, Tænia solium, Tænia saginata, Diphylobothrium latum*; some pass from man to man, or from animals to man by way of crustacea or fish—*Fasciolopsis buskii, Clonorchis sinensis, Heterophyes heterophyes, Paragonimus ringeri, Schistosoma hæmatobium et mansoni*. The Filariidæ are transmitted by biting insects.

Some of these helminths are rare, others are common; two are common in certain areas and may be the cause of serious disability—*Ancylostomum duodenale* and *Schistosoma hæmatobium et mansoni*—the former on account of the anæmia it may cause, the latter as being responsible for urinary and intestinal schistosomiasis, which is pandemic in the Nile area. Recently attention has been drawn to the frequency with which the cysticercus stage of *Tænia solium* may give rise to an incurable form of epilepsy.

All these helminth infestations are preventible—by the control of foods, the effective disposal of fæces and urine, the prevention of wading and bathing in infested water, the control of mosquitoes—so that a knowledge of these worms and of their life histories may enable an officer to anticipate and prevent cases from occurring. By early diagnosis, through the identification of eggs or larvæ in the excretions, or by skin reactions to extracts of the infesting species, early treatment may prevent chronic invalidism. Particularly is this the case with schistosome infections, for which antimony and emetine are both virtually specific.

Finally, a few words concerning venereal disease may not be out of place. In Australia these complaints seem to be relatively rare, and I have been surprised at the interest created by the exhibition of such a trivial sight as a primary chancre. In the East venereal disease is, of course, pandemic among the class of prostitute with whom troops may be expected to consort, and it is certain that numbers of men will become involved whatever precautions are put at their disposal.

Gonorrhœa is ubiquitous in the East, and it is fortunate that sulphapyridine has now altered the outlook in this condition. I have, however, often been surprised at the casual manner in which this drug is exhibited, and take leave to doubt many of the statistics emanating from out-patient clinics. Gonorrhœa is a serious infection, and once the prostate is involved can be regarded as practically incurable. Patients must be made to present themselves immediately symptoms appear, they must be at once put to bed and given a very low diet with plenty of alkaline fluid, and sulphapyridine, one gramme every four hours, must be administered until the patient is evidently intoxicated by the drug. The dose is then reduced, but the treatment is maintained for a further four or five days. Given these conditions, gonococci should vanish in thirty-six hours and never reappear, reinfection apart.

Syphilis is also extremely common in the East. When conditions are difficult, the advisability of giving a concentrated course of arsenic ("Novarsenobillon" or "Mapharside") may be considered—a full dose every other day for five doses, the subject being given large amounts of glucose throughout the procedure (Chevalier).

Soft sores must be distinguished from chancres, and it seems to be sometimes forgotten that in the diagnosis of syphilis dark-ground illumination of serum from sores is well regarded as more useful and more scientific than the Wassermann reaction. For Ducrey infections "Dmelcos" and "M & B 693" are both valuable in treatment.

Poradenitis venerea is common in all eastern ports. Enlargement of the inguinal glands with fever and general malaise is also typical of plague—a point worth remembering. Frei antigen is valuable in the diagnosis of poradenitis. This condition is often known as *lymphogranuloma inguinale*, and is sometimes confused with *granuloma venereum*, the rarest venereal infection—a spreading and destructive ulceration of the external genitalia associated with Leishman-Donovan bodies.

Unfortunately, such is the filthy condition of oriental prostitutes, that multiple infections are common among men who have hired them, to say nothing of the crab lice that are probably also present. This multiplicity of

infections is not peculiar to venereal disease in the tropics, and the physician must on that account be constantly on his guard against accepting one so-called specific ætiological factor. Amœbic and bacillary dysentery often coexist, as do malaria and enteric fever; while ankylostomiasis may cooperate with any other chronic infection to reduce a subject to a serious condition of invalidism. The failure of specific treatments is commonly to be accounted for in the overlooking of multiple infections.

Conclusion.

I have purposely ranged over a considerable field, to create the impression that the medical officer in the tropics under conditions of war has a hard task. I have understated rather than over-stated the case. Unless he can win the wholehearted cooperation of his men, unless he carries in his head a wealth of factual knowledge, unless he can apply that knowledge with the greatest possible efficiency under every disadvantageous condition, he will fail in his duty to a greater or lesser degree. Upon his success or failure, as much as upon that of those who determine major and local strategy, may depend the outcome of the contest.

Acknowledgements.

I am indebted to Dr. A. Neave Kingsbury, Director of the Institute for Medical Research, for permission to publish this paper.

The following authorities have been responsible for text-books containing important factual knowledge concerning the prevention, diagnosis and treatment of disease in the tropics: General text-books: Byam and Archibald, Castellani and Chalmers, de Langden and Lichtenstein, Manson-Bahr, Stitt, Rogers and Megaw. Bacteriology, Protozoology, Helminthology: Acton and Knowles, Blacklock and Southwell, Chandler, Stitt and Clugh, Topley and Wilson, Wenyon.

SOME ASPECTS OF TWO COMMON TROPICAL DISEASES.[1]

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THE selection of material for this paper has been somewhat difficult, because in the time at my disposal it will be possible only to touch briefly on a few points of interest in one or two of the more important tropical diseases. I have therefore decided that it will be of more interest and value to my audience if I discuss certain aspects of the diseases which are the most likely to be acquired by large numbers of our troops at present serving in the Middle East. I trust that this will meet with your approval and that the information I am about to give you will not be regarded as of too elementary a nature, particularly by those of you who have had some experience of tropical medicine.

Malaria.

You will not be surprised if I begin with malaria, because in the last war it was responsible for more casualties than any other disease in that portion of the British Army which served in countries bordering on the eastern Mediterranean and in East Africa. So far as my information goes, and I must admit it is meagre, there has been little if any invaliding of Australian troops abroad on account of malaria yet; but if it does make its appearance in our army, it is practically certain that it will be in a severe and fatal form similar to that seen in the last war. The following are some of the reasons for this dangerous probability: (i) There is a large and intimate concentration of men who have never suffered from malaria and hence are highly susceptible. (ii) The concentration of so many men gives numerous opportunities of rapid transmission of the disease from man to man, and the short sojourn of the parasite in the mosquito will probably lead to its enhanced virulence. (iii) Although the men are undoubtedly in excellent training and are a specially selected body, they are living under conditions of heavy mental and physical strain, which will no doubt have an influence in lowering their resistance.

Those medical officers who are serving with the troops in the field will be concerned with the treatment and management of the acute primary attack; but those of you who will be concerned with treating men who have had relapses and have been invalided from the front to Australia, and who probably comprise the majority of this gathering, will be much more interested in this aspect of the problem. Therefore, instead of trying to give a full account of the treatment of this disease, I propose to confine my remarks to a few of the main points regarding the principal new synthetic drugs that have been introduced and used with considerable success in the treatment of malaria since the last war.

First I shall give a short account of the clinical appearances you are likely to encounter. It is sure to be the exception rather than the rule that the usual text-book description of malaria will be seen in your "relapse" cases (that is, the regular sharp rise of temperature preceded by a rigor and followed by sweating, with complete intermission of the abnormal temperature for one or two days between the attacks, according to the type of infection that is present). It is much more probable that you will find a temperature which steadily mounts from day to day, with only partial and indefinite remissions and perhaps slight feelings of chill, but no definite rigor, also at irregular intervals. Pain, headache *et cetera* are common, but in no way characteristic, and the only clinical sign of real value is a large, tender and hard spleen, which is practically always present in uncured malaria. The probable reason for this irregularity in the clinical signs is that after a time some parasites because of a lag, and others because of a speeding-up in the completion of their cycle, gradually grow further and further apart, so that eventually numbers of red cells are bursting and liberating merozoites more or less continuously; this has the effect of keeping the fever going all the time. This consideration indicates that the only certain means of rapid diagnosis of malaria is examination of a blood film, and if thick blood films are used parasites are usually readily found even before they have increased to any great extent.

Up till about 1926, in spite of prolonged experiment and trial, no drug but quinine was proved of any real value in the treatment of malaria, and it was frequently quoted as one of the best examples of a drug specific for a disease. Although considerable success has attended the use of the drugs I shall mention in a few minutes, they have by no means yet supplanted quinine as still the most powerful remedial agent against malaria we possess, because the supply of the new drugs is strictly limited, and they are even more expensive than quinine, which is by no means a cheap drug.

Like the majority of efficient chemotherapeutic agents that have been synthesized in the laboratory, the original source of these drugs was Germany, and until the outbreak of the war that country was still the main source of supply. We are in a much better position this time than at the outbreak of the last war to replace these and other similar manufactured drugs, because both British and American chemists are now able to prepare drugs either identical with German preparations or quite efficient substitutes for them.

The first of the synthetic drugs of use in malaria was "Plasmoquine" (or "Plasmochin"). It is a dye derivative with methylene blue as its point of origin, and is actually fairly closely allied to quinine. It is found to be useful in the treatment of benign tertian and quartan malaria, as it has a rapidly destructive action on all stages and forms of these parasites as they occur in the blood. It is peculiar, however, in that it has no action on the asexual forms of the malignant tertian parasite, but has been found to be the best agent at our disposal for destroying

[1] Read at a meeting of the Victorian Branch of the British Medical Association on February 5, 1941, at Melbourne.

the sexual forms (crescents) by which the disease is transmitted through the mosquito. It is a toxic drug causing cyanosis, severe abdominal pain and vomiting, so it must be administered only under medical supervision and the dose for an adult should never exceed 0·06 gramme per day for one week. It is a useful drug combined with quinine, as less of the latter is needed to effect a cure than when it is given alone, and it is available in the form of "Plasmoquine compound" tablets which contain 0·01 gramme of "Plasmoquine" and 0·125 gramme of quinine, or as "Quinoplasmine" containing 0·01 gramme of "Plasmoquine" and 0·3 gramme of quinine. But probably its greatest value is in the pure form "Plasmoquine simplex", which is put up in 0·01 or 0·02 gramme tablets and can be used to eradicate the sexual forms of malignant tertian malaria which often persist in the blood after all the asexual forms have been banished. Its value in this way is to prevent mosquitoes from picking up the infection from persons apparently cured of an attack of malignant tertian malaria, and thus it is a useful agent in checking the spread of an epidemic.

On account of the limitations of "Plasmoquine" as a cure for malaria, further research was carried out, and about six years after its introduction another drug, also based on the methylene blue molecule, was produced. This is known as "Atebrin", or as the Americans term it, "Atabrin". It is much less toxic than 'Plasmoquine" and is effective against all species and all forms of the three malaria parasites except the gametocytes of malignant tertian. It causes complete disappearance of the parasites from the blood in two to three days in the average case, and it is claimed by several workers of repute to effect a complete cure in nearly every case. This has not been the experience of my colleagues in Calcutta, however, for relapses have been frequently found to occur there, and although it is not possible to exclude the chance that some of these cases are due to reinfection, it seems clear that many of them have been true relapses.

The ordinary course of "Atebrin" is 0·1 gramme three times a day for five to seven days. It is rapidly absorbed from the intestine, and relatively slowly excreted from the body by the kidneys and in the bile. Both quinine and "Plasmoquine" are much more rapidly eliminated. "Atebrin" can also be used intravenously, and given in this way it has a great advantage over quinine; it remains in the blood stream for several days, whereas quinine disappears so rapidly that for it to be effective in severe cases, injections have often to be repeated several times, whereas a single injection of "Atebrin" will usually control a heavy infection. It was extensively used by the intravenous method in the great Ceylon epidemic about four years ago, where it was found to be of particular value in the treatment of children.

"Atebrin" and "Plasmoquine" have also been used in combination, and although the mixture is valuable, as one would expect, it is not recommended as a routine measure because the toxic effects of "Plasmoquine" appear to be enhanced in this way. It is probably better to give a course of "Atebrin" first and then to follow it with "Plasmoquine" for the reasons I have indicated above.

"Atebrin" is considered as a favourable prophylactic, and in doses of 0·1 gramme per day can be taken for months without the production of any objectionable symptoms. Investigations into this aspect of the use of "Atebrin" are not complete; but as far as they go, they indicate that it is probably slightly better than 0·3 gramme (five grains) of quinine, the usual prophylactic daily dose.

"Atebrin" can be used in blackwater fever with safety, for persons who suffer from quinine hæmoglobinuria, and for pregnant women, as it has no action on the uterus; thus it is a very useful alternative to quinine in any of these circumstances. It is much less toxic than "Plasmoquine" and can be given in double the efficient therapeutic dose without the production of any symptoms; in spite of its slow elimination it causes no signs of cumulative effects if an average course of five to seven days' treatment is repeated after a few days' interval. A common and alarming sign to the uninitiated is a yellow staining of the skin and conjunctivæ. This is not a sign of liver involvement, but only staining by the drug itself, indicating that some of it is being eliminated by this route.

"Tebetren" is a combination of hydroquinine, an acridine dye and a bile salt. It is of considerable value in the treatment of malaria, but as far as Calcutta experience goes it is not considered to be so good as "Atebrin".

Research is still proceeding, or was doing so until the outbreak of the war, and many other drugs have been produced and hailed as useful antimalarials. It is not possible to mention these, except to say that so far as one is able to determine none of them has proved so good as "Atebrin".

Sulphanilamide preparations have been tried for malaria as they have for practically every other disease, and although they have a distinct effect on the parasites, in their present form they are not so good as quinine, "Atebrin" *et cetera*.

Amœbic Dysentery.

I shall next say something about amœbic dysentery, as there are sure to be many carriers of this infection among the returned men, and once again I shall not discuss the primary acute attack, but shall confine my remarks to the carriers and the usual signs of this infection found in them.

In the beginning I should like to stress that care must be used in describing these persons, not as "cyst carriers", but as carriers of *Entamœba histolytica*, since the former term indicates ignorance of the true condition present. Cysts of this organism in the stools always means that living amœbæ are present somewhere in the intestinal tract or its adnexa. Some authorities (notably Craig in America, who has had enormous experience of this disease in central American countries) hold that even in the complete absence of symptoms in carriers there are always actual lesions in the gut, however minute they may be, in which the vegetative or active stage of the parasite is living and multiplying, ready at any time to set up an active diseased condition.

One of the most important characteristics of this infection in the chronic state is that a person may carry it for years without exhibiting any symptoms whatever, and apart from being a danger to his fellows, is likely at any time to become acutely ill by a flare-up in the activity of the amœbæ. The cause of this exacerbation is often impossible to determine, but dietetic indiscretions and chill seem to be important predisposing factors. As a digression I shall mention the fact that persons who have this infection are very susceptible to chill in the abdomen, and anyone who habitually suffers from colic after moderate degrees of chill in this area of his body should always be regarded with suspicion as a possible carrier of *Entamœba histolytica* or some other chronic intestinal infection.

One of the most striking cases of unsuspected infection of this nature, with no previous symptoms and a sudden onset of a very serious condition, that has come within my personal experience, is the following:

A colleague of mine in England with whom I was in close touch was apparently in perfect health, when one morning in his bathroom he collapsed with severe hæmorrhage into the bowel. Immediate operation saved him, and it was found that the cause of the crisis was ulceration into a large enough artery in his intestinal wall to endanger his life. Subsequently X-ray examination showed that practically the whole large bowel was honeycombed with ulcers, which were shown to be of amœbic origin. Total removal of the colon was performed; he recovered, and after fifteen years is still doing valuable research work.

I quote this case, not because you are likely to encounter a parallel, but to emphasize what can occur as a result of an unrecognized amœbic infection, and I have only to leave it to your own imagination to visualize the endless possibilities that may arise.

Chronic amœbiasis may be classed in two main types: (i) that in which the pathological changes are situated in the intestine, (ii) that in which the liver is the site of the disease. The first type may present an endless

variety which it is not possible to describe in detail; but the main varieties can be classified in the following three ways: (*a*) There may be a sudden attack of typical acute dysentery, with the passage of blood, mucus and numerous active amœbæ. (*b*) There may be vague and irregularly occurring attacks of abdominal pain not typical of any particular disease, which in the absence of positive findings on examination of the stools may suggest chronic appendicitis, cholecystitis *et cetera*, but which is revealed in its true light by the presence in the stools of the typical cysts. (*c*) There may be no symptoms at all.

An acute relapse is, of course, treated in the same way as an initial acute attack, and the best drug to use is emetine given by injection. One grain a day for nine to twelve days constitutes a full course; this amount should not be exceeded, because it is a toxic drug and tends to accumulate in the tissues. Animal experiments, vouched for by Dale, have proved that overdosage causes gastro-enteritis, acute nephritis, pulmonary œdema and muscular weakness. *Post mortem*, liver changes and a pale, flabby heart are found in addition. On account of its early effect on the heart patients should always be made to go to bed during a course of treatment, and on account of its slow elimination from the body a second course is not advisable in less than about three months. It is true that many people have taken emetine while going about their usual work, but numerous accidents have followed this practice and it is better avoided.

Emetine is not very effective in chronic cases in which there are few or no symptoms, so apart from the objection that the drug makes it necessary for an apparently healthy person to go to bed for about a fortnight, it is not much used in this type of case. In these cases many drugs have been recommended and many of them appear to be fairly effective; but all of them fail in a good proportion of cases, so the true specific is still to be found. I shall not have time to do more than enumerate some of the best of them.

Several of these drugs contain emetine or ipecacuanha. "Alcresta Ipecec." is an adsorption compound of the total alkaloids of ipecacuanha with fuller's earth. A few of the drugs most often used are emetine-bismuthous-iodide, emetine periodide (relatively non-toxic), auremetine (a combination of emetine and the dye auramine), arsemetine (a definite chemical combination of arsenic and emetine) and "Gavano" (a Bayer product the full chemical formula of which has not been divulged).

The bark and seeds of a plant known as *Holarrhena antidysenterica* (*Kurchi* in Bengali) is a remedy for dysentery that is recorded in ancient Indian medicine. It has been fully studied both chemically and pharmacologically at the Calcutta School of Tropical Medicine, and the powdered bark, the individual alkaloids, the total alkaloids in combination called *Kurchi*-bismuthous-iodide, have been tried extensively; it appears that the drug is about equal in effect to emetine and ipecacuanha. It is one of the few valuable drugs that have been found out of the great number of those mentioned in ancient Indian writings, which have been tested at the Calcutta School to see whether there was any scientific basis for their early reputation.

Many organic arsenicals, such as "Stovarsol" and "Carbarsone", have also been used with some success, as well as mercurials.

The position regarding these chronic carriers is obviously unsatisfactory, and one of the reasons for the doubt as to the value of these numerous remedies is the difficulty in establishing the fact that a cure has been effected. Many of the drugs, if they do not completely eradicate the infection, reduce it to such an extent for the time being that repeated examination of the stools fails to reveal the presence of cysts. Thus a cure is claimed; but later the few remaining amœbæ multiply, with the result that the condition again becomes obvious microscopically and also perhaps clinically.

Let me say a few words on the finding of cysts in the stools. It is considered that one examination will reveal 75% of infections, two examinations will reveal 90% and six examinations will reveal nearly 100%. There are occasional cases, especially those in which the amount of infection has been greatly reduced by treatment, and in these as many as twelve examinations have been known to fail to demonstrate the infection.

These figures indicate that it would probably be worth while in after years to examine once and preferably twice the stools of all patients who show on their history sheets that they have ever had this infection whenever they again come under medical supervision, no matter what the cause may be. I admit that routine examinations of all hospital patients by all departments are tedious and time-consuming; but in Calcutta where this is done there is ample evidence that in the long run it well repays the effort, both in the acquisition of valuable statistical data and in the shortening of the time of treatment in many cases.

Liver Involvement.

I shall now consider the other important form of chronic amœbiasis, liver involvement. In this again we find all stages and degrees of severity, from mild hepatitis with no ascertainable enlargement of the liver and no pain or tenderness, merging into definite enlargement and tenderness of the liver. The next type appears much the same; but in this there are numerous small abscesses scattered throughout the organ. Finally there are cases in which one or more definite large localized abscesses cause distortion and displacement, the amount of which depends on the situation of the abscess and its nearness to the liver surface.

Diagnosis is difficult, particularly in the simple hepatitis in which there is no rise of temperature, no pain and no tenderness. It very largely depends on the previous history of amœbic infection and the possible presence of cysts in the stools. Often the only positive sign is a leucocytosis with a great increase in polymorphonuclear cells; but this is, of course, by no means pathognomonic. If a definite localized abscess is not present, it is not safe nor is it worth while to try to aspirate any material for diagnosis. If this is done when an abscess is present, one must not expect to find active amœbæ in the typical anchovy sauce-like material that the abscess contains, because they live only in the abscess walls and rapidly die and degenerate when they are cast off into the cavity. All that can be said of X-ray examination is that it is sometimes of help.

Without going into further detail than this in the clinical diagnosis of amœbic liver abscess, which you will gather from the above remarks would be not of much practical value, I think the position may be summed up in the teaching of one of the honorary surgeons at the Royal Melbourne Hospital in my day (I think it was Fred Bird). In discussing inert abdominal tumours, particularly those of the liver, he said that in this part of Australia at least, one should never forget that the lesion might be a hydatid cyst. This may be translated into the following words: when persons come from centres where amœbic dysentery is endemic and exhibit signs of liver disease, always consider amœbiasis among the possibilities.

Early diagnosis is important, because these patients respond in a remarkable manner to a course of emetine injections, and so abscess formation may be aborted. Even after a definite abscess has formed emetine may cause its absorption; therefore after the diagnosis is made it is worth while trying this method first before resorting to aspiration or to the much more serious open operation.

As a corollary to these observations on the recognition of amœbic abscess of the liver and its successful treatment with emetine, it is interesting to note that so-called idiopathic "tropical liver", once commonly diagnosed in those who returned home after years spent in the tropics, is now practically unknown.

Conclusion.

I trust that the fact that I have confined my remarks to two common diseases and to only part of them will not have disappointed you. I felt that if I tried to cover a wide field and to touch on many diseases the references would of necessity have been too brief to convey a clear

impression of anything, and that you would have left the meeting with a feeling that you had been told little of value. Finally, I shall give a warning, the need for which I assure you is not without foundation in my experience. It is that in the tropics we get all the diseases of temperate climates as well as those peculiar to warm countries. Therefore if a returned soldier comes into your care and the disease from which he is suffering is not immediately apparent, do not think that because he has been in Egypt or elsewhere abroad he must necessarily be suffering from some obscure tropical complaint, but give the home diseases consideration while making your diagnosis.

TSUTSUGAMUSHI FEVER IN NORTH QUEENSLAND, AUSTRALIA.

By W. G. Heaslip.

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Although tsutsugamushi fever was not definitely diagnosed in Queensland until 1927 or later by Paine and Nye (Langan and Mathew, 1935), Cilento (1923) had previously pointed out the similarity between certain of the Queensland fevers and Japanese river fever. Clarke (1913) gave the first recognizable description of the condition. He states that the earliest white settlers on the Daintree River, north of Mossman, suffered severely from fever, many cases being fatal; also that the fever was known amongst the natives before the arrival of the settlers in 1877.

The present studies were begun in 1938, as part of a general investigation into the undiagnosed fevers of North Queensland, at the invitation of Dr. E. H. Derrick, Director of the Laboratory of Pathology and Microbiology, Brisbane. For this purpose Dr. Derrick had been given a financial grant by the National Health and Medical Research Council.[1]

I have previously described the separation of cases of tsutsugamushi[2] from other similar cases of fever (Heaslip, 1940) and the organism, since named *Bacillus tropicus*, associated with both. In that paper is given a full list of previous publications concerning tsutsugamushi in Australia. There, also, I have acknowledged the assistance given and the courtesy shown to me by a number of people. Dr. C. Leggett, the present superintendent of the Cairns District Hospital, extended to me the same privileges and courtesy as his predecessor. Further assistance in the shape of clinical data was supplied by Dr. M. L. Unwin, Tully; Dr. H. S. Harper, Mossman, Dr. R. A. D. Pope, Cooktown; Dr. R. S. Cohen, Gordonvale; and Dr. T. W. Carroll, Babinda.

Lewthwaite and Savoor (1940) have concluded from the results of their extensive observations and experiments that Sumatran mite fever, scrub typhus and tsutsugamushi are one and the same disease, and that the two former names should therefore be discarded. Although I have not been able to carry out cross-immunity tests, there seems to be no reason why the condition discussed in this paper should not also be identified with tsutsugamushi.

While tsutsugamushi as a name has been associated with this particular fever in Japan for many years, it simply means "dangerous bug". Although the "bug" has been proved a mite, and we thus have the name "dangerous mite fever", this is not a satisfactory denomination. It is probable that the mite is capable of transmitting organisms other than *Rickettsia tsutsugamushi*, and the name gives no indication of the fact that the disease belongs to the typhus group of fevers. At present the most suitable name appears to be "K typhus".

[1] Subsequent grants from the Council enabled this work to be continued without interruption.

[2] Throughout the text the name "tsutsugamushi fever" is contracted to "tsutsugamushi".

The Causal Agent.

The organism responsible for the disease has been given a variety of names. Hayashi (1920) claimed to have seen it in blood smears and tissue cells, and called it *Theileria tsutsugamushi*. Later, Hayashi (*Tropical Diseases Bulletin*, Volume XXXI, 1934, page 252) claimed that he described and named the organism *Rickettsia tsutsugamushi* in 1922. In 1923 Sellards gave the name *Rickettsia nipponica* to an organism which he cultivated on chocolate agar. This was neither a *Rickettsia* nor the causative organism of tsutsugamushi. Nagayo *et alii* (1931) called the organism *Rickettsia orientalis*; Ogata (1931) named it *Rickettsia tsutsugamushi*; and Kawamura and Imagawa (1931) named it *Rickettsia akamushi*. In this paper it will be designated *Rickettsia tsutsugamushi*.

The rickettsiæ seen in the present investigations correspond with those described previously. In addition it was found that they might survive for at least three days in blood at room temperature (about 15° to 30° C.), for eight days but not nine days in pleural fluid at −10° C., and for five minutes in blood and pleural fluid at 60° C. Infective pleural fluid was mixed with an equal quantity of a 1% solution of phenol just prior to injection into mice, which died in the usual time from tsutsugamushi. The association of the organisms with the disease, and of the infection in mice with that in man, was demonstrated by the occurrence of infection in a laboratory assistant. At the time he was working in Adelaide, where natural infection is unknown (see below).

For routine staining, to demonstrate the organism, carbol-thionin prepared according to the method of Laigret and Auburtin (1938) was used. This stain is cheap, quick and effective. With it the organism is usually seen in the bipolar form. Although usually found in the cytoplasm of endothelial cells, it may be seen in white cells. Smears from freshly killed infected mice occasionally show the organism in blood corpuscles and free in tissue fluids.

Tsutsugamushi in Man.

My observations in Cairns occupied fifteen months, covering a period of two years and including two seasons of maximum incidence. Fifty-four cases were investigated and the details are set out in Table I.

Mortality Rate.

Only one of the 54 cases was fatal—a mortality rate of 2%. The patient who died was elderly and was not admitted to hospital until the evening before death occurred. It is possible that with adequate care he might have survived. Mathew (1938) gives a total of 71 cases diagnosed in three years, with four deaths, a mortality rate of 5·6%. This figure is high, as probably all the fatal cases for the three years, but only a proportion of the actual cases of tsutsugamushi, were included in the series. Even had the serum from every suspected case been examined some cases would still have been missed, as many patients leave hospital before agglutinins appear. Wheatland (1924) gives the mortality rate in the Sarina outbreak as 20% to 30%. Clarke (1913) states that many cases among the early settlers were fatal.

Clinical Features.

In the paper mentioned above I pointed out that in most cases there was a double infection (see also Table I), and that when the bacillary infection only occurred, the clinical condition might be indistinguishable from that in cases of double infection. Consequently one cannot say definitely what is the clinical picture of tsutsugamushi in Queensland. The fact that what I observed corresponds closely with the findings in other countries may mean (i) that the superimposed infection has little effect on the clinical condition, or (ii) that the double infection occurs in other countries also. It is probable that the extended periods of pyrexia, which are frequently noticed, are due to the double infection.

In addition to the data given in Table I, most patients complain of headache, anorexia and weakness, and exhibit furred tongue, conjunctivitis, adenitis and considerable

TABLE I.

Showing the Results of the Investigation of Fifty-four Cases of Tsutsugamushi.

Patient.	Age. (Yrs.)	Description, Source and Locality.	Date of Onset.	Date of Admission to Hospital.	Duration of Fever.	Spleen.	Eschar.	Rash.	Mice.	Blood Collected.	Agglutination of Proteus OXK by Serum.	Remarks.
F.McC.	24	Lives in West Cairns.	13/10/38	17/10/38	Days. 9+5	N.p.[1]	N.s.[2]	N.s.	N.u.[3]	8/11/38 21/11/38	1 in 1280 1 in 320	Not diagnosed until readmitted to hospital on 7/11/38. No attempt at culture.
J.S.	18	Getting wood, Double Island swamp.	11/11/38	18/11/38	23	P.[4]	N.s.	Appeared 22/11/38	N.u.	22/11/38 30/11/38 1/12/38	N.a.[5] 1 in 320 1 in 640	Long pyrexial period probably due to double infection. No culture, but lymphocytosis.
G.B.	34	Farmer's wife, Babinda.	22/11/38	24/11/38	19	N.p.	N.s.	Present.	Died.[7]	29/11/38 7/12/38	N.a. 1 in 2560	Double infection in the mice.
W.B.	23	Timber - getting, Tully.	21/11/38	23/11/38	10	N.p.	Left pectoral region.	Present.	N.u.	6/12/38	1 in 320	No blood culture.
J.McC.	42	Mining in scrub, Edmonton.	10/12/38	19/12/38	14+7	N.p.	N.s.	Appeared 15/12/38	Died. One died, other two subsequently immune.	20/12/38[10] 26/12/38 29/12/38[10] 2/1/39 6/1/39	N.a. 1 in 80 1 in 2560 1 in 1280 1 in 640	Double infection in mice. On 22/12/38 developed pneumonia. Proteus OX19 agglutination titre rose from nil to one in 160. (See text.)
V.E.	32	Timber cutting, Mourilyan swamp.	18/12/38	28/12/38	12	N.p.	N.s.	Present.	Died.	29/12/38[10] 2/1/39	N.a. 1 in 2560	Double infection in mice. Partner of E.C. below.
E.C.	20	Timber cutting, Mourilyan swamp.	18/12/38	26/12/38	13	N.p.	N.s.	N.s.	Died.	28/12/38 30/12/38[10]	1 in 80 1 in 640	Double infection in mice. Same camp as V.E. above.
J.W.	18	Timber cutting, El Arish.	21/12/38	23/12/38	11+3	N.p.	Lower left part of abdomen.	Present.	N.u.	3/1/39 11/1/39	1 in 640 1 in 320	No blood culture.
E.R.	44	Road construction, camp, Jordan Creek.	28/12/38	4/1/39	9	N.p.	N.s.	Present.	N.u.	5/1/39 11/1/39	N.a. 1 in 20	No blood culture. No further serum available.
J.C.	51	Working in scrub, El Arish.	13/2/39	14/2/39	14+3	P.	Abdomen.	Indefinite.	N.u.	13/3/39	1 in 160	Double infection.
W.C.	46	Lives in West Cairns.	28/2/39	3/3/39	11+6	N.p.	N.s.	Appeared 6/3/39	Died. (Guinea - pig given one cubic centimetre clot, died of bacillary infection within 48 hours.)	6/3/39[10] 9/3/39 (O.S.[11] 17/3/39 (O.S. 20/3/39 23/3/39	N.a. N.a. 1 in 80) 1 in 640 1 in 5120) 1 in 1280 1 in 5120	Double infection. *Bacillus tropicus* seen in direct blood smears. "Prontosil" given without obvious effect.
H.L.	14	Farmhand, Cairns Intake.	28/2/39	3/3/39	10+9	N.p.	N.s.	N.s.	N.u.	10/3/39 14/3/39 20/3/39 (O.S.	1 in 160 1 in 160 1 in 160 1 in 320)	Aborigine. Disease clinically typical. *Bacillus tropicus* in blood culture.
A.B.	27	Timber - getting, Edmonton.	18/3/39	22/3/39	8	N.d.[8]	N.d.	N.d.	None died. Two of three subsequently immune.	23/3/39 28/3/39[10]	1 in 20 1 in 640	No growth in attempts at blood culture.
W.H.	32	Lives in West Cairns.	27/3/39	3/4/39	9	P.	N.s.	N.s.	Did not die. Had enlarged spleens.	5/4/39[10] 11/4/39	1 in 1280 1 in 640	Double infection. Early appearance of agglutinins.
W.R.	34	Lives in West Cairns.	5/4/39	12/4/39	8	N.p.	N.s.	Present.	N.u.	13/4/39 19/4/39	1 in 160 1 in 160	Typical clinically. *Bacillus tropicus* in blood culture. Proteus OX19 agglutination nil and one in 80 respectively.
W.G.	40	Working at landslide, Cairns Intake.	7/4/39	14/4/39	12+3	P.	N.s.	Present.	N.u.	15/4/39 26/4/39	1 in 80 1 in 10,240	Double infection. Same source as three following cases.

[1] N.p. = Not palpable.
[2] N.s. = Not seen.
[3] N.u. = Not used.
[4] P. = Palpable.
[5] N.a. = No agglutination.
[6] N.d. = No data.
[7] Died = Death due to rickettsial infection.
[8] Male.
[9] Female.
[10] Indicates sample of blood injected into mice.
[11] O.S. = Oxford Standard emulsion of Proteus OXK.

TABLE I.—*Continued.*

Showing the Results of the Investigation of Fifty-four Cases of Tsutsugamushi.—Continued.

Patient.	Age. (Yrs.)	Description, Source and Locality.	Date of Onset.	Date of Admission to Hospital.	Duration of Fever.	Spleen.	Eschar.	Rash.	Mice.	Blood Collected.	Agglutination of Proteus OXK by Serum.	Remarks.
S.S.	41	Working at landslide, Cairns Intake.	9/4/39	17/4/39	Days. 10+6	N.p.	Abdomen.	N.s.	Died.	19/4/39[17] 26/4/39 1/5/39 17/5/39	1 in 40 1 in 160 1 in 640 1 in 320	Double infection. Same source as three others.
E.G.	32	Working at Landslide, Cairns Intake.	18/4/39	22/4/39	9	N.p.	N.s.	Appeared 24/4/39	N.u.	24/4/39 1/5/39 16/5/39	1 in 20 1 in 320 1 in 640	Double infection. Same source as three others.
E.T.	46	Working at landslide, Cairns Intake.	20/4/39 Preceded by six days' malaise.	24/4/39	14	N.p.	Abdomen.	Present.	Died.	26/4/39[19] 2/5/39 8/5/39 14/5/39	1 in 320 1 in 640 1 in 1280 1 in 640	Double infection. Same source as three preceding cases.
W.G.	32	Timber - getting, Little Mulgrave.	3/4/39	N.d.	21 or more.	N.d.	N.d.	N.d.	N.u.	24/4/39 2/5/39	1 in 320 1 in 640	Blood culture not attempted.
J.McC.	29	N.d., Gordonvale.	29/4/39	N.d.	17	N.d.	N.d.	N.d.	N.u.	8/5/39 16/5/39	N.a. 1 in 1280	Blood culture: growth of undetermined organism.
A.C.	30	Getting wood, Edmonton.	22/4/39	28/4/39	11+5	N.p.	Abdomen.	Present.	Did not die; spleen enlarged. The same, second passage fatal.	1/5/39[18] 5/5/39[18] 9/5/39 14/5/39	1 in 20 1 in 40 1 in 40 1 in 40	Double infection.
J.H.	52	Slaughterman, Queerah.	30/4/39	4/5/39	9	N.p.	N.s.	N.s.	(1) Mice did not die, spleens only moderately enlarged. (2) Guinea pigs passage then to mice, which died.	5/5/39 9/5/39[18] 18/5/39	N.a. 1 in 10 1 in 20	Double infection.
P.C.	12	Farmer's son, Redlynch.	7/5/39	9/5/39	12	N.p.	Left costal margin posteriorly.	Indefinite.	Killed 22/5/39, spleens greatly enlarged.	10/5/39[18] 15/5/39 19/5/39	N.a. 1 in 320 1 in 1280	Double infection. One mouse died within 48 hours of bacillary infection.
K.P.[2]	25	Lives at survey camp, Mossman.	18/9/39	25/9/39	12+9	N.p.	N.s.	Present.	N.u.	2/10/39 6/10/39	1 in 20 1 in 160	No blood culture. Definite history of tick bite. Three other cases from same camp.
G.W.	17	Farmhand, Daintree.	4/10/39	8/10/39	9	P.	N.s.	Present.	Died.	16/10/39	1 in 320	Double infection.
H.W.	30	Surveyor, Mossman.	5/10/39	9/10/39	9	P.	N.s.	Present.	Died.	16/10/19[18] 23/10/39	N.a. N.a.	Double infection. History of tick bite. See K.P. above.
C.W.	25	Timber - getting, Edgehill.	16/10/39 Malaise for preceding four days.	18/10/39	8	N.p.	N.s.	Appeared 19/10/39	Did not die.	20/10/39[18] 25/10/39 28/10/39 31/10/39	N.a. 1 in 20 1 in 20 1 in 10	*Bacillus tropicus* in blood culture. Typical clinically, but not proven.
Geo.W.	21	Surveyor, Mossman.	16/10/39	18/10/39	7+	N.p.	N.s.	N.s.	Died.	19/10/39[18] 4/11/39	N.a. 1 in 640	Double infection. History of tick bite. See H.W. and K.P. above.
C.P.	28	Miner in scrub, Cooktown.	13/3/40	23/3/40	16	N.p.	N.s.	N.s.	N.u.	23/3/40 29/5/40	1 in 40 N.a.	Double infection, probably. *Bacillus tropicus* in blood.
J.O.	39	Timber - getting, Cairns Intake.	22/3/40	30/3/40	31	P.	Abdomen.	N.s.	One died of bacillary infection, two died. Died.	1/4/40[18] 8/4/40[18] 12/4/40 17/4/40	N.a. 1 in 20 1 in 160 1 in 1280	Double infection. Gland puncture; smears: no organisms seen; culture: *Bacillus tropicus*.
L.R.	15	Working in scrub near Ingham.	25/3/40	3/4/40	10+5	P.	Left axilla.	N.s.	Died.	3/4/40[18] 8/4/40 12/4/40 17/4/40	N.a. 1 in 80 1 in 640 1 in 1280	Double infection. Gland puncture; smears: occasional organism; culture: *Bacillus tropicus*.

TABLE I.—*Continued.*

Showing the Results of the Investigation of Fifty-four Cases of Tsutsugamushi.—Continued.

Patient.	Age. (Yrs.)	Description, Source and Locality.	Date of Onset.	Date of Admission to Hospital.	Duration of Fever.	Spleen.	Eschar.	Rash.	Mice.	Blood Collected.	Agglutination of Proteus OXK by Serum.	Remarks.
N.F.	23	Grubbing stumps, Meerawa.	17/4/40	23/4/40	Days. 10	N.p.	Left groin.	Present.	Died.	26/4/40	N.a.	Double infection. Only one sample of serum received.
L.P.	21	Farmhand, Little Mulgrave.	4/5/40	6/5/40	9	P.	N.s.	N.s.	Died.	7/5/40	N.a.	Not diagnosed clinically. Double infection.
T.McL.	33	Mining in scrub, Cooktown.	15/4/40	22/4/40	10+2	N.p.	N.s.	N.s.	N.u.	2/5/40 13/5/40	1 in 40 1 in 10	*Bacillus tropicus* in blood culture. Doubtful case.
H.C.	44	Working in scrub, Mossman.	Two weeks' malaise.	10/5/40	12+	N.p.	Leg.	N.s.	N.u.	25/5/40	1 in 1280	Doubtful infection.
T.M.	41	Working near swamp, Mossman.	7/5/40	11/5/40	11+7	N.d.	Abdomen.	N.s.	N.u.	24/5/40	1 in 160	Doubtful infection.
S.H.	42	Works at survey camp, Mossman.	8/5/40	12/5/40	15	N.d.	N.d.	Present.	N.u.	24/5/40 30/5/40	1 in 20 1 in 40	Typical clinically. *Bacillus tropicus* in in blood culture.
Ar.C.	28	Timber - getting, West Cairns.	14/5/40	21/5/40	11+7	N.p.	N.s.	Present.	One of two died.	15/5/40 27/5/40[10] 4/6/40	N.a. 1 in 20 1 in 320	Double infection.
M.Z.	52	Clearing swampy scrub, Queerah.	13/5/40	20/5/40	13+9	N.p.	N.s.	N.s.	One died bacillary infection. Other died in 25 days. spleen greatly enlarged. Lungs infected.	22/5/40	N.a.	Doubtful case, typical clinically. Further serum not available. Gland puncture. Culture : *Bacillus tropicus*.
M.W.	58	Fisherman's wife. Camped in bush, Yorkie's Knob.	11/5/40	16/5/40	15	N.p.	N.s.	N.s.	N.u. till 1/6/40; did not die.	21/5/40 29/5/40 1/6/40	N.a. 1 in 40 1 in 160	Double infection.
D.S.[8]	6	Two weeks camped in scrub, Tully.	17/5/40	22/5/40	9	P.	N.s.	Appeared 22/5/40	N.u.	28/5/40	1 in 2560	Double infection.
A.M.	45	Timber - getting, Cooktown.	13/5/40	16/5/40	16	N.p.	N.s.	Present.	N.u.	23/5/40 6/6/40	N.a. 1 in 20	Doubtful case. *Bacillus tropicus* in blood culture.
H.V.	41	Timber - getting, Double Island.	21/5/40	5/6/40	20	N.p.	N.s.	Appeared 26/5/40	One died, other subsequently not immune.	6/6/40 12/6/40	1 in 40 1 in 1280	Double infection. Gland puncture. Culture : *Bacillus tropicus*.
P.B.	19	Farmhand, West Cairns.	29/5/40	7/6/40	14	P.	Abdomen.	Indefinite.	N.u.	8/6/40 13/6/40 23/6/40	N.a. Indefinite. 1 in 80	Double infection.
T.P.	25	Farmer, near Tully.	2/6/40	6/6/40	10+3	P.	N.d.	Appeared 8/6/40	One died, other had enlarged spleen.	12/6/40	1 in 160	Double infection. Blood three days old when injected into mice.
A.L.	26	Timber - getting, Little Mulgrave.	16/6/40	N.d.	N.d.	N.d.	N.d.	N.d.	N.u.	15/7/40	1 in 5120	Double infection. See I.L. (wife) below.
V.C.	23	Timber - getting, West Cairns.	25/6/40	30/6/40	10	N.p.	Right flank.	Appeared 30/6/40	One only died. Subsequently the strain died out in mice.	2/7/40[10] 9/7/40	1 in 20 1 in 40	Double infection. Gland puncture. Culture : *Bacillus tropicus*. Same locality as Ar.C. above.
H.K.	49	Timber - getting, El Arish.	26/6/40	30/6/40	11	N.P.	Left thigh.	Present.	N.u.	10/7/40	1 in 160	Blood incubation : no growth.
D.B.	54	Working in West Cairns.	30/6/40	10/7/40	Died 11/7/40	N.p.	Left axilla.	N.d.	Died.	10/7/40	1 in 40	Double infection.
S.L.	43	Lives in West Cairns.	14/7/40	19/7/40	11+3	N.p.	Right axilla.	N.s.	Died.	19/7/40	N.a.	Double infection.
V.A.[8]	11	Lives in West Cairns.	Malaise from 1/8/40	Not admitted to hospital; in bed from 11/8/40	Afebrile 21/8/40	N.p.	N.s.	Appeared 11/8/40	One died; the other had a greatly enlarged spleen.	20/8/40[10] 27/8/40 3/9/40	1 in 160 1 in 640 1 in 640	Double infection. Originally diagnosed as measles. Had similar attack one year previously.

TABLE I.—*Continued.*

Showing the Results of the Investigation of Fifty-four Cases of Tsutsugamushi.—Continued.

Patient.	Age. (Yrs.)	Description, Source and Locality.	Date of Onset.	Date of Admission to Hospital.	Duration of Fever.	Spleen.	Eschar.	Rash.	Mice.	Blood Collected.	Agglutination of Proteus OXK by Serum.	Remarks.
E.L.	17	General carrier, Cairns District.	17/8/40	26/8/40	Days. 12+4	N.p.	N.s.	Appeared 21/8/40	Did not die; one had inapparent infection. Second passage mice died.	26/8/40[10] 30/8/40 7/9/40 14/9/40	N.a. N.a. N.a. N.a.	Double infection. Rickettsia in smears from first passage mouse.
I.L.	26	Wife of timber-getter living in scrub, Little Mulgrave.	29/8/40	17/8/40	9+3	N.p.	N.s.	N.s.	N.n.	5/9/40 13/9/40	1 in 40 1 in 640	Double infection. Wife of A.L. above. In hospital twelve days before onset.

TABLE II.

Giving the Results of the Case of Laboratory Infection with Tsutsugamushi.

Date.	Inoculated Mice.	Attempts at Blood Culture for Presence of *Bacillus tropicus*.	Agglutination of Proteus OXK.	Erythrocyte Count per Cubic Millimetre.	Leucocyte Count.	Differential Count.[1]				
						Polymorphonuclear Cells.	Lymphocytes.	Monocytes.	Eosinophile Cells.	Plasma Cells.
23/1/40	All died.	——	No agglutination.	—	—	—	—	—	—	—
25/1/40	—	Type-form bacilli present at 18 hours. None at 24 hours or subsequently.	No agglutination.	6,180,000	4,200	66	14	20	—	—
26/1/40	All died.	——	——	—	—	—	—	—	—	—
27/1/40	—	No growth.	1 in 20	5,820,000	6,300	75	10	15	—	—
29/1/40	All died.	Bipolar form grew in clot.	No agglutination.	—	5,000	65	12	23	—	—
31/1/40	—	No growth.	No agglutination.	5,680,000	6,900	72	16	10	1	1
2/2/40	2 died—typical. 2 not affected.	No growth.	1 in 20	—	6,250	49	31	19	2	—
4/2/40	—	No growth.	No agglutination.	—	—	—	—	—	—	—
6/2/40	None affected.	No growth.	No agglutination.	5,000,000	6,900	59	26	14	1	—
8/2/40	None affected.	Late growth of bipolar form.	Partial at 1 in 20, 1 in 40 and 1 in 80	—	—	—	—	—	—	—
10/2/40	None affected.	Bipolar bacilli.	1 in 80	—	10,200	58	32	10	—	—
12/2/40	—	Bipolar bacilli.	1 in 160	—	—	—	—	—	—	—
14/2/40	None affected.	No growth.	1 in 320	—	12,000	58	32	6	4	—
16/2/40	—	No growth.	1 in 320	—	—	—	—	—	—	—
19/2/40	—	No growth.	1 in 160	5,900,000	9,000	64	22	9	5	—
26/2/40	—	No growth.	1 in 80	—	—	—	—	—	—	—

[1] These counts were kindly made by Dr. J. B. Thiersch, Neale Research Pathologist, at The Institute of Medical and Veterinary Science, Adelaide.

loss of weight. Less constant symptoms are generalized aches and pains, sore throat with or without cough, nausea with or without vomiting, and sore eyes. Occasionally disturbances of sensation are reported. Variable signs are injected fauces, enlarged tonsils, tenderness of lymphatic glands, liver or spleen, enlargement of spleen or liver, and photophobia. The spleen was palpable in only 12 of 48 cases. In severe cases delirium may occur.

There are great variations in the degrees of pyrexia, but frequently there is a fall of temperature after about ten days. In Table I this is indicated by the duration of pyrexia being given in two periods.

Complications.—Complications are largely confined to pulmonary troubles; bronchitis is frequent, and pneumonia occasionally supervenes. In the fatal case jaundice was present and the Van den Bergh reaction was biphasic. Abdominal distension accompanied by pain was prominent in one case, and priapism lasting one week occurred in a boy aged eleven years. Pulmonary conditions, jaundice and abdominal pain all occur in cases of bacillary infection alone.

Diagnosis.—If an eschar is present the diagnosis is apparent; without an eschar it may be exceedingly difficult, and without laboratory aids is nearly always doubtful. A rash was present in 29 of 47 cases; when it is typical and occurs about five days after the onset it is of considerable aid in diagnosis. Lewthwaite and Savoor (1940) give four criteria of infection in monkeys: eschar, leucopenia, febrile reaction and agglutination of Proteus OXK; but they add: "Not each of the four criteria of infection may develop, and differences in degree of reaction are common." In Queensland patients rarely have a leucopenia. This is possibly due to the lymphocytosis which develops in cases of bacillary infection. An eschar was observed in only 17 of 48 cases; in some cases it may have been present without being recorded. However, it was present in only 12 of the 35 patients whom I examined myself. Frequently the patient leaves hospital before agglutinins develop, and in one case no agglutinins had developed twenty-eight days after the onset. Inoculation into white mice gave good results; but with this method also the patient may have left hospital before the diagnosis is established. For statistical purposes, when an eschar is not present it is necessary to make both serological tests and animal inoculations. A few doubtful cases have been included in Table I to emphasize the difficulty of diagnosis.

Incubation Period.—In a few instances exposure to infection was limited to a definite period. In one it was not more than ten days; in another it was not more than fifteen and not less than eleven days. The patient who died was exposed to infection for a few hours ten days before the onset. He was exposed again from the sixth to the third day before the onset. The patient M.L., who was the wife of the patient A.L., was admitted to the Gordonvale Hospital with another complaint twelve days before the onset of tsutsugamushi.

Source of Infection.

Possible vectors are discussed below, also the proved reservoirs. From Table I it appears that practically all the patients lived or worked in proximity to scrub or swampy areas. The known animal reservoirs are not limited to these areas, but mites and ticks are, more particularly the former.

Serology.

Blood was collected from patients and the serum was tested for agglutinins of Proteus OXK and Proteus OX19 as frequently as was expedient or possible. For various reasons complete serological examination was achieved in only a few cases. The results are set out in Table I. The diversity of reaction is well illustrated by these results, and it is obvious that usually a rise or fall of titre must be demonstrated before a significant result is established.

The emulsion used was kindly provided by the Director of the School of Public Health and Tropical Medicine, Sydney. It was compared with an Oxford standard emulsion, but an agglutinability factor was not determined, as it varied with the serum from different patients. However, the former emulsion was considerably less agglutinable than the latter. This probably explains some of the low titres obtained, and possibly also why no agglutinins were found in the serum of the patient E.L. On the other hand, it does not explain why there was no alteration of titre twenty days after the onset of illness in Case 12 (H.L.), nor why this patient had an apparently permanent high-level titre. It is possible that the titre eventually rose in this instance, as agglutinins sometimes first appear more than twenty days after the onset. On the other hand, in the serum of patient W.H. the titre was 1 in 1,280 after only nine days, and had fallen to 1 in 640 by the fifteenth day. These results illustrate again the danger of attempting diagnosis on one serum examination. In two instances there was an apparently permanent titre of 1 in 160, while in some definite cases of typhus the maximum titre did not reach this level. (See also the case described below.) The blood may still be infective for mice when the agglutination titre has reached a high level.

Significant agglutination of Proteus OX19 was found in only two instances (patients J.McC. and W.R.). In the former the titre rose from nil on December 20, 1938, to 1 in 40 on December 26, 1938, and to 1 in 160 on January 6, 1939; and in the latter it rose from nil on April 13, 1939, to 1 in 80 on April 19, 1939. The onset was on December 10, 1938, and on April 5, 1939, respectively, and the rise of titre was not typical of endemic typhus in either case, since it usually begins earlier and progresses more rapidly.

The serum of a patient proved to be suffering from leptospirosis agglutinated Proteus OXK at 1 in 40 on May 4, 1939. On May 11, 1939, the titre had risen to 1 in 160. Inoculated mice had only moderately enlarged spleens. This was probably a non-specific rise of titre.

A Laboratory Infection.

In addition to the cases listed above, an assistant became ill on January 20, 1940.

The patient had been handling infective mouse material at intervals of two or three days for a fortnight before the onset, and on January 15, 1940, had spent an entire morning dissecting infected mice and making serial passages.

On examination when the illness started he was found to have a small abrasion on the outer aspect of the second knuckle of his right forefinger. From this a lymphangitis could be traced to the right elbow. The right axillary glands were enlarged and slightly tender. A typical rash appeared on the fifth day and the pyrexial period lasted for eleven days. General adenitis developed, but only the glands in the right axilla were much enlarged. A comprehensive investigation of his blood was made and the results are set out in Table II.

The mice were given citrated blood, usually 0·3 cubic centimetre, intraperitoneally. On the occasion when two died and two did not, the former were quite typical in every way, and the latter, killed later, at no time showed any sign of infection.

The positive agglutination results on January 27, 1940, and February 2, 1940, were confirmed by repeated testing. The partial agglutination obtained on February 8, 1940, when there was no complete agglutination, has been observed several times. This occurs only at the onset of typical production of agglutinins.

Broth was the medium in which the type-form of *Bacillus tropicus* was temporarily present on January 25, 1940; the organisms were present in a stained smear and in a hanging-drop preparation. Although no organisms grew in the several instances up to February 6, 1940, their presence in the blood was demonstrated by subsequent investigation of the inoculated mice and by inoculation of the blood into the yolk sac of developing hen's eggs.

There were an early mild leucopenia, with an increase of monocytes, and a late lymphocytosis, relative and absolute.

The patient developed a cough in the second week of illness, which persisted for some time after his recovery. He complained only of nausea when he thought about eating, and of an evanescent, throbbing headache following movement.

The rash faded rapidly after the second day and had disappeared three days after it appeared. The patient weighed 155 pounds when admitted to hospital and 141 pounds when he was discharged; by that time he had regained some weight.

Animal Experiments.

White Mice.

I reported previously (Heaslip, 1940) that white mice were found susceptible to the disease in Queensland. Mice were first used successfully by Dinger (1933) in Java, and later by Wolff and Kouwenaar (1934) in Sumatra. The last authors reported almost 100% mortality rate. Okamoto (1936) found pleurisy and peritonitis in mice infected with "Manchurian mild endemic typhus", and saw *Rickettsia* in cells of serous membranes, of alveoli of the lungs, of the splenic venous plexus and of hepatic veins. Ogata (1936) also successfully used mice.

On the other hand, Tanaka *et alii* (1930) state that mice are resistant to Japanese river fever, and most other workers in Japan appear to have had the same experience. The same apparently is true in Malaya, and Lewthwaite and Savoor (1940) recently reported that when they received a Sumatran strain in mice it survived for three passages only. Apparently some strains of white mice are resistant and some susceptible to infection.

While all my mice were not susceptible to the Queensland infection, mouse inoculation has proved the best diagnostic method. With an Oxford standard suspension of Proteus OXK, serum agglutination might prove equally successful.

The pathological findings in infected mice were essentially the same as those described by Dinger (1933). Usually there were both pleural and peritoneal effusion, but occasionally neither was present. Wolff and Kouwenaar (1934) rarely saw either. However, they did observe constant splenic enlargement, which is the important finding. My records of several hundred observations show a very constant relation between spleen and body weight. The normal spleen is less than 0·25% of body weight. Infections with *Bacillus tropicus* may cause enlargement up to 0·5% of body weight. If the spleen weight exceeds 0·5% of the weight of the inoculated mouse, then tsutsugamushi infection should be suspected. Even when the presence of an abscess at the site of inoculation or elsewhere offers an explanation of the gross enlargement, it is wise to make a further passage.

For original inoculations from man or animals with active infection, citrated blood has proved to be the best inoculum. For passages, if citrated blood is not available, or if the infection is dormant, an emulsion of liver in citrated saline solution is best. Fluid from the pleural cavity is rather less reliable. When infection with *Bacillus tropicus* occurs, the use of solid organs for passage material should be avoided. The blood is infective from the fourth day onwards, possibly earlier.

Death results usually in ten or eleven days, but may occur as early as four days or as late as twenty-one days, possibly later. A recently acquired human strain killed the first mice in fourteen days. The second-passage animals died after nine days. The third passage animals (three) were exposed to low night temperatures while being taken from Cairns to Brisbane by ship, and died after six, seven and eight days respectively. No individual in any of the succeeding nine passage groups has survived for more than nine days.

One strain was kept in mice by serial passage for forty-four weeks and then voluntarily discarded.

Guinea-Pigs.

Guinea-pigs could be infected, but death resulted or signs of infection appeared only occasionally. In one instance a strain of *Rickettsia tsutsugamushi* survived in them for fourteen weeks (seven passages) but then ceased to be manifest. Mice were used to detect the presence of infection at each passage. Rapid loss of weight is the most obvious indication of infection.

Rabbits.

I obtained the same results with rabbits as workers elsewhere, except that eye inoculations were sometimes complicated by the multiplication of *Bacillus tropicus* in the anterior chamber. Large quantities of inoculum given into the peritoneal cavity were necessary to produce significant agglutination titres. Rabbits can be successfully used for the production of experimental eschars.

Monkeys.

The serum from five normal monkeys (*Macacus mulatta*) was tested for agglutination of Proteus OXK. Two specimens gave complete agglutination at a titre of 1 in 10, three at a titre of 1 in 20 and one at a titre of 1 in 40.

In addition the following tests were made.

1. A female *Macacus mulatta* monkey was bled and the serum was tested with Proteus OXK, which it agglutinated at a titre of 1 in 20. On September 1, 1939, the animal was given pleural fluid from an infected mouse, 0·1 cubic centimetre intradermally, and 1·2 cubic centimetres into the peritoneal cavity. A typical eschar developed, with accompanying adenitis. There was mild pyrexia from September 3, 1939, to September 14, 1939. The agglutination titre rose to 1 in 40 on September 19, 1939, and on December 4, 1939, it was 1 in 20.
2. A male *Macacus sinicus* monkey was tested and the serum was found to agglutinate Proteus OXK at a titre of 1 in 20. On September 1, 1939, the animal was inoculated with fluid from an infected mouse; 0·1 cubic centimetre intradermally and 1·2 cubic centimetres intraperitoneally. Induration and hyperæmia, which did not progress to eschar formation, resulted. General adenitis followed initial local adenitis. The period of pyrexia was seven days. On September 19, 1939, the serum titre was 1 in 80; on October 6, 1939, it was 1 in 20. On November 30, 1939, the monkey was given measles virus intranasally, and on December 4, 1939, the serum titre was 1 in 80. This supports the suggestion that a non-specific rise of titre may occur.
3. A male monkey, *Macacus cynomolgus*, was tested and the serum agglutinated Proteus OXK at a titre of 1 in 40. On December 22, 1939, it was given 0·5 cubic centimetre of citrated blood from an infected mouse into and under the skin. No eschar developed, though hyperæmia and induration appeared at the site of injection. Adenitis followed and the pyrexial period was ten days. No rise in serum titre was observed. On December 29, 1939, blood was withdrawn and given to three white mice, and on January 5, 1940, the same procedure was carried out. Both batches of mice subsequently died with typical lesions, and rickettsiæ were observed in smears. The monkey subsequently died of tuberculosis without showing any rise in titre.

Rats.

In an early attempt to define the possible reservoir of tsutsugamushi several species of rats were inoculated with material from infected mice, with the following results:

Rattus norvegicus, white strain: Two rats were used, and both became infected but survived.

Rattus norvegicus, black and white strain: Two rats were used, and both became infected but survived.

Rattus conatus: Two were used, both became infected and died under an anæsthetic.

Rattus culmorum: Two were used, both became infected and died under an anæsthetic; one had pleurisy and peritonitis with effusion.

Rattus assimilis: One was used, became infected and died under an anæsthetic; the post-mortem findings were the same as in mice.

Melomys littoralis: Two were used, one escaped and one had an inapparent infection.

All these species except *Rattus culmorum*, and in addition *Rattus rattus*, were found later to be naturally infected. In each case the infection was proved by mouse inoculation.

Bandicoots.

Only one species of bandicoot, *Isoodon torosus*, appears to be present around Cairns. It was found to be capable of harbouring infection after inoculation, and was later shown to be a natural reservoir. In the case of both rats and bandicoots, the blood of the test animals, taken before inoculation, was injected into mice to exclude the presence of other infections. The results obtained in the bandicoots are set out in Table III.

Serum collected from 82 bandicoots at the Laboratory of Pathology and Microbiology, Brisbane, were tested for agglutination of Proteus OXK and Proteus OX19. Despite the fact that all these animals came from southern Queensland, where no cases of tsutsugamushi have been reported, 24 specimens of serum gave agglutination of Proteus OXK in titres up to 1 in 80. None of them agglutinated Proteus OX19.

TABLE III.

Showing the Result of Artificially Infecting Bandicoots (Isoodon torosus) with Tsutsugamushi.

Bandicoot.	Place of Origin.	Serum.	Test Mice.	Bandicoot Inoculated.	Blood Withdrawn and Given to Mice.	Result.
67	Nambour, South Queensland.	1/2/39 OXK 1/80 8/2/39 OXK 1/80	No infection.	1/2/39 with 0·5 cubic centimetre blood and pleural fluid.	8/2/39	Mice died of tsutsugamushi.
68	Nambour, South Queensland.	1/2/39 N.a.[1] 8/2/39 N.a. 14/2/39 OXK 1/40 15/4/39 OXK 1/40	No infection.	1/2/39 with 0·5 cubic centimetre blood and pleural fluid.	8/2/39 14/2/39	Mice died of tsutsugamushi. Not infected.
72	Cowan Cowan, South Queensland.	2/2/39 N.a. 9/2/39 N.a. 16/2/39 OXK 1/20	No infection.	2/2/39 with 0·6 cubic centimetre liver emulsion.	9/2/39 16/2/39	Mice died of tsutsugamushi. Mice died of tsutsugamushi.
79	Cairns, North Queensland.	1/7/40 N.a.	No infection.	1/7/40 with 5·0 cubic centimetres liver emultion.	11/7/38 29/7/40 19/8/40	Mice infected, but did not die. Mice died of tsutsugamushi. Mice not infected.
80	Cairns, North Queensland.	16/5/40 N.a.	No infection.	21/5/40 with 5·0 cubic centimetres liver emulsion.	31/5/40 12/6/40 2/7/40 (killed and liver emulsion given to mice).	Mice infected, but did not die. Mice not infected. Mice infected, but did not die until second passage.

[1] N.a. = No agglutination.

Developing Hen's Eggs.

Infective blood or pleural fluid, or both, was inoculated into the yolk sac of developing hen's eggs at various times by the method described by Cox (1938). All attempts at serial passage in eggs failed because of infection with *Bacillus tropicus*, which killed the embryos. However, on one occasion second-passage eggs proved fatal for mice, which died typically of tsutsugamushi. Rickettsiæ were present in smears from these mice.

Some of these experiments were carried out at the Commonwealth Health Laboratory, Cairns, some at the Laboratory of Pathology and Microbiology, Brisbane, and some at the Institute of Medical and Veterinary Science, Adelaide.

The Reservoir.

When it had been demonstrated that the local species of rats and bandicoots could be infected with tsutsugamushi, an endeavour was made to prove natural infection in these animals. Trapping was carried out during 1939 and again in 1940. Only live animals were used.

All animals were killed with chloroform and blood was collected by heart puncture under aseptic conditions. The serum was tested for agglutinins of Proteus OXK and Proteus OX19. Smears were made from the pleura, peritoneum, spleen and *tunica vaginalis*. The viscera were removed and the carcass was kept for the collection of ectoparasites. If there was any doubt about identification of the animal, the carcass was preserved and later sent to the Australian Museum, Sydney, where the identification was kindly checked by the mammalogist, Mr. E. L. Troughton. It is noteworthy that in 195 rats there was no specimen of *Rattus culmorum*. In 1939 only occasional mouse inoculations were made, but in 1940 a liver emulsion from most of the animals was injected intraperitoneally into a batch of white mice. As a routine measure, attempts at blood culture were made to determine the presence of *Bacillus tropicus*.

The results are set out in Table IV. An extended examination of the smears from the animals trapped in 1940 was not made. However, those from the 80 caught in 1939 were searched for rickettsiæ with great care. Apparently the organisms are only sparsely present at times other than that of acute infection. The division of the infections into definite, probable and possible was based on the demonstration of rickettsiæ, on the occurrence of death at the usual time with post-mortem appearances of tsutsugamushi, and on the presence of splenic enlargement, respectively, in one or more of the inoculated mice. It is likely that the number of definite and probable infections would have been more, and the possible infections correspondingly less, had all the surviving

TABLE IV.

Showing the Results of Certain Investigations of Captured Rats and Bandicoots.

	Rats.		Bandicoots.	
	Number Examined.	Result.	Number Examined.	Result.
Smears	195	*Rickettsia* seen in only one (*Rattus conatus*).	54	No *Rickettsia* seen.
Specimens of serum ..	195	51 agglutinated Proteus OXK at titres up to 1 in 320. 10 agglutinated Proteus OX19 at titres up to 1 in 80. No apparent correlation with active infection.	49	13 agglutinated Proteus OXK at titres up to 1 in 80. 3 agglutinated Proteus OX19 at titres up to 1 in 40. No apparent correlation with active infection.
Blood incubation for culture	183	*Bacillus tropicus* grew in 119 instances. Of the 64 samples yielding no growth, 26 were found to contain *Bacillus tropicus* in the course of experiments with mice.	54	*Bacillus tropicus* grew in 37 instances. Of the 17 samples that yielded no growth, three were found to contain *Bacillus tropicus* in the course of experiments with mice.
Natural infection with tsutsugamushi tested by mouse inoculation.	125	15 definite. 9 probable. 15 possible.	52	1 definite. 7 probable. 8 possible.

TABLE V.
Showing Infection Rate in Various Species of Rats from Two Types of Locality.

Species.	Bush.					Swamp.				
	Tested.	Infected.	Definite.	Probable.	Possible.	Tested.	Infected.	Definite.	Probable.	Possible.
Rattus conatus	15	4	3	1	—	1	—	—	—	—
Rattus rattus	10	2	1	1	—	52	26	8	7	11
Rattus assimilis	7	1	1	—	—	—	—	—	—	—
Rattus norvegicus	1	—	—	—	—	2	1	1	—	—
Melomys littoralis	27	2	1	—	1	10	3	—	—	3
Total	60	9	6	2	1	65	30	9	7	14

inoculated mice been killed on the twelfth to the fourteenth day, and passages made whenever tsutsugamushi infection was suspected. This would have given the mice time to die of the infection and would have avoided the danger (i) of the infection's dying out, and (ii) of using liver emulsion for passage, which sometimes leads to death from infection with *Bacillus tropicus*. Because this was not reached until late in the investigation and for other reasons passage was sometimes as late as five weeks after inoculation (see protocols).

Further details regarding the species of captured rats and the natural infection rate are set out in Table V. Although a similar number of rats was caught in the bush as in the swamp, the trapping period was at least twice as long in the bush. The bush areas were some miles from Cairns and the swamp area in which the trapping was done was actually in West Cairns. *Rattus norvegicus* was represented by only three specimens, and the specimens of *Rattus rattus* taken in the bush were caught near farmhouses.

The higher proportion of definite to total infections in the bush rats is probably due to the fact that they were caught from two to eight weeks earlier (May and June) than the swamp rats; the infection had probably passed the acute stages in many of the latter when they were captured.

The weight of the spleen and the body weight of each rat were recorded; in the possibly, probably and definitely infected groups the spleen weight represented 0·29%, 0·32% and 0·31% of the body weight respectively. In the 58 bush rats, untested or not found infected with typhus, the spleen averaged 0·21% of the body weight.

Protocols relating to mouse inoculations from two of the possibly infected rats were as follows:

Rat 1 was killed on May 30, 1940. An emulsion of the liver was injected into M.403 (two mice). On May 31, 1940, one died of bacillary infection. On July 2, 1940, the other was killed; its weight was 19·5 grammes and the spleen weighed 250 milligrammes. An emulsion of the liver was injected into M.456 (two mice). On July 24, 1940, one was dead (injured); its weight was 20 grammes and the spleen weighed 80 milligrammes. The other mouse was killed; it weighed 22 grammes and its spleen 220 milligrammes. A liver emulsion from the latter was injected into M.503 (three mice). On July 26, 1940, two were dead from bacillary infection. Their weights were 20 and 19 grammes respectively and the spleens weighed 90 and 70 milligrammes respectively. On August 12, 1940, one was killed. It was pregnant and weighed 33 grammes; the spleen weighed 170 milligrammes. A liver emulsion was injected into M.597 (two mice). On September 4, 1940, both mice were killed; they weighed 26 and 23 grammes respectively and their spleens weighed 110 and 90 milligrammes respectively.

Rat 4 was killed on July 16, 1940, and a liver emulsion was injected into M.471 (one mouse). On August 13, 1940, it was killed; it weighed 19 grammes and its spleen 190 milligrammes. A liver emulsion was injected into M.605 (two mice). On August 19, 1940, one was dead; it weighed 11 grammes and its spleen 100 milligrammes. A liver emulsion was injected into M.609 (two mice). On September 5, 1940, the other M.605 was killed; it weighed 23 grammes and its spleen 80 milligrammes. On August 20, 1940, both M.609 mice were dead of bacillary infection; their weights were 20 and 18 grammes respectively and the spleens weighed 70 and 60 milligrammes respectively.

In addition to the species of rat found infected (see Table V), some specimens of *Hydromys chrysogaster regina* and *Uromys caudimaculatus* were captured and found to harbour larval mites.

Bandicoots (*Isoodon torosus*) were captured and treated in the same way as the rats. Of 52 tested, one was definitely infected, seven were probably infected and eight were possibly infected. The details relating to the definitely and probably infected bandicoots are set out in Table VI.

The ectoparasites removed from the animals are discussed below. It needs to be emphasized that a clump of larval trombids removed from an animal (usually from the region of the anus in bandicoots and from the ears of rats) very seldom consisted of only one species.

The Vector.

It has frequently been stated that *Trombicula akamushi* Brumpt 1910 has been proved the vector of tsutsugamushi in Japan, and there is sufficient evidence to incriminate larval mites beyond reasonable doubt. However, since the larvæ normally feed only once, and after moulting are probably strict vegetarians, it is necessary for absolute proof of specific vectorship (i) to breed the larvæ from a definitely identified adult, and (ii) to produce the disease by allowing the known larvæ to attack susceptible animals. I can find no record that this has been done in Japan or elsewhere. It has also been stated that Walch and Keukenschrijver (1924) proved *Trombicula deliensis* Walch 1922 to be the vector in the Dutch East Indies. Actually only a very suspicious association was demonstrated.

In Queensland the epidemiology of the disease is such that only mites and ticks need be considered as vectors. It is scarcely possible that a tick could commonly be the vector without being seen and recognized. On the other hand, four cases occurred at a survey camp near Mossman, in each of which there were a definite history of tick bite and an equally definite denial of mite infestation. I have explained earlier (Heaslip, 1940) why a denial of mite infestation is worthless as evidence; but the possibility remains that a tick could be the vector occasionally.

In addition to the usual epidemiological evidence of mite vectorship, the following facts are available:

1. On three occasions an emulsion of larval mites was inoculated into different batches of white mice. None of the animals died from infection, but they were eventually killed. In one batch there was splenic enlargement compatible with tsutsugamushi infection.

2. About 90% of some 2,500 larval mites, removed from captured animals, were specimens of *Trombicula deliensis*. I agree with Gater (1930 and 1932) that *Trombicula akamushi* and *Trombicula deliensis* are at most variants of the same species (see also Fletcher *et alii*, 1928).[1] Hatori (1919) in Formosa, and Walch (1923) in Sumatra claim

[1] Mr. H. Womersley, entomologist to the South Australian Museum, who has specialized in acarology, agrees with this opinion. I am indebted to him for instruction, assistance, advice and encouragement. He has checked the identification of all the mites mentioned in the section on ectoparasites.

TABLE VI.

Showing the Results of the Examination of Eight Bandicoots (Isoodon torosus), Seven Probably and One Definitely Infected with Rickettsia Pathogenic for White Mice.

Series Number.	Locality.	Body Weight. (Grammes.)	Weight of Spleen. (Grammes.)	Blood Incubation for Culture.	Agglutination of Proteus OXK and OX19 by Serum.	Results of Intraperitoneal Injection of Suspected Material into White Mice.[1]
81	Bush near Cairns (Intake).	—	—	No growth. Mice infected with bacilli.	No agglutination.	16/5/40 citrated heart blood. M.[3]385 → 13/6/40. 1 killed; B.,[2] 18 grammes; S.,[4] 110 milligrammes. / 4/6/40. 1 dead; B., 13 grammes; S., 180 milligrammes. Clinically typhus → [5]M.420 → 5/6/40. 1 dead; bacillary infection. / 10/7/40. 1 killed; B., 22 grammes; S., 190 milligrammes. / 11/7/40. Given human strain typhus material; killed 1/8/40; B., 22 grammes; S., 240 milligrammes.
82	Bush near Cairns (Intake).	—	—	Bacilli.	OX19. No agglutination. OXK +1/80	20/5/40 citrated heart blood. M.386 → 13/6/40. 1 killed; B., 20 grammes; S., 130 milligrammes. / 9/6/40. 1 dead; B., 17 grammes; S., 120 milligrammes; clinically typhus.
89	Bush near Cairns (Intake).	870	2·18	Bacilli.	OX19 no agglutination. OXK ±1/80	27/5/40 citrated heart blood. M.398 → 29/5/40. 1 dead; B., 19 grammes; S., 60 milligrammes. / 6/6/40. 1 dead; B., 16 grammes; S., 100 milligrammes; clinically typhus →M.430. 7/6/40. All dead; bacillary infection.
100	Swampy area in Cairns.	1,300	2·80	Bacilli.	No agglutination.	18/7/40. M.477. 21/8/40. Killed; B., 22 grammes; S., 220 milligrammes→ →M.621. 4/9/40. 2 dead; B., 16 and 15 grammes; S., 120 and 100 milligrammes; *Rickettsia* seen in smears.
112	Swampy area in Cairns.	250	1·40	No growth. Mice infected with bacilli.	No agglutination.	25/7/40. M.513 → 21/8/40. 1 dead; B., 18 grammes; S., 240 milligrammes; clinically typhus. / 22/8/40. 2 killed; B., 22 and 19 grammes; S., 120 and 170 milligrammes→ →M.624 6/9/40. Killed; B., 17 and 18 grammes; S., 60 and 70 milligrammes.
114	Swampy area in Cairns.	800	3·20	Bacilli.	No agglutination.	28/7/40. M.521 → 20/8/40. 1 dead; B., 18 grammes; S., 140 milligrammes; clinically typhus. / 21/8/40. 2 killed; B., 22 and 27 grammes; S., 160 and 200 milligrammes→ →M.619 5/9/40. Killed; B., 21 and 20 grammes; S., 80 and 80 milligrammes.
118	Swampy area in Cairns.	840	2·90	Bacilli.	No agglutination.	29/7/40. M.533 → 14/8/40. 1 dead; B., 22 grammes; S., 240 milligrammes; clinically typhus. / 15/8/40. 2 killed; B., 20 and 23 grammes; S., 100 and 130 milligrammes→ →M.607 5/9/40. Killed; B., 25 and 28 grammes; S., 100 and 260 milligrammes→ →M.655 23/9/40. Killed; B., 21 grammes; S., 70 milligrammes.
119	Swampy area in Cairns.	400	0·72	No growth. Bacilli in inoculated mice.	No agglutination.	30/7/40. M.545 30/8/40. Killed; B., 25, 24 and 22 grammes; S., 80, 150 and 180 milligrammes→ →M.645 → 3/9/40. 1 dead; bacilli present. / 13/9/40. 1 dead; B., 8 grammes; S., 70 milligrammes; clinically typhus.

[1] Unless otherwise stated, the mice were inoculated with liver emulsion. [2] M. = Batch of white mice. [3] B. = Body weight. [4] S. = Spleen weight. [5] → indicates "passaged to".

to have bred the same nymph, *Trombicula coarctata* Berlese 1912, from *Trombicula akamushi* and *Trombicula deliensis* respectively.

3. Gunther (1940), seeking the vector of tsutsugamushi in New Guinea, found what he thought was a local variant of *Trombicula deliensis* and named it *Trombicula vanderghinstei*. Mehta (1937) states that *Trombicula deliensis* is found in association with scrub typhus in the Simla Hills, India. In short, this mite seems to be closely associated geographically with tsutsugamushi infection.

It is therefore probable that *Trombicula akamushi* (*Trombicula deliensis*) is the usual vector in Queensland and elsewhere.

On only one occasion was I able to collect larval mites from a human host. The species taken was *Trombicula hirsti* Sambon 1927. Infestation occurred at Lake Barrine, about 1,500 feet above sea level, and this species was not found on rats or bandicoots caught at or near sea level. Gunther (1939), who collected 3,000 mites from animals and birds in the Bulolo Basin, New Guinea, states that 2,700 of them were specimens of *Trombicula hirsti*. They were not found on rats, and the majority came from birds, though some were taken from bandicoots and man. Bulolo is about 4,000 feet above sea level. On the other hand, from these larvæ Gunther bred nymphs which apparently belong to the species *Trombicula minor* Berlese 1904, and I found adult specimens of *Trombicula minor* at or near sea level in Queensland. Gunther (1938) suggested that in New Guinea *Trombicula hirsti* was the vector of tsutsugamushi and that its infrequent occurrence on the supposed reservoir, the bandicoot, explained the sporadic nature of the cases there. However, it is possible that *Trombicula deliensis* (*Trombicula vanderghinstei*) is the vector; it is found there on rats, the usual reservoir, and its sparse distribution in the Bulolo Basin would also explain the infrequent occurrence of the disease. Moreover, *Trombicula hirsti* appears not to occur in association with tsutsugamushi in Japan and India. Further, there is evidence that other larval mites cause far less irritation than *Trombicula hirsti*, and this may be the real explanation of the frequent lack of history of mite infestation in cases of tsutsugamushi.

Ectoparasites.

The long-standing confusion with regard to the classification of the Trombidiidæ is growing with the list of members. This is due largely to amateur acarologists who do not appreciate the difficulties of identification, partly to unsatisfactory drawings and descriptions, partly to the inaccessibility of the literature, and partly to a misguided enthusiasm for creating new species and variants. I mention this solely because acarology has become important medically, and there is an urgent need for a central institute where identification of specimens can be checked and satisfactory breeding experiments carried out.

I examined more than 2,500 larval trombids taken from some 200 captured animals, and it was rarely that an animal was attacked by only one species. Not less than 14 species representing five genera were found attached to rats and bandicoots. In addition to *Trombicula minor* (*Trombicula hirsti*) found on man and *Trombicula deliensis* mentioned above, there were specimens of an unidentified, probably new species of *Trombicula*; two specimens of an unidentified species of either *Trombicula* or *Neoschöngastia* (the sensilli were missing in both); specimens of *Neoschöngastia derricki* Womersley 1939; of a new, similar species and a variant; *Neoschöngastia indica* Hirst 1915; a species resembling *Neoschöngastia coorongense* Hirst 1929; one specimen which resembled *Neoschöngastia westraliense* Womersley 1934; and three other unidentified, probably new species of *Neoschöngastia*, one having the base of each dorsal seta in the centre of a round platelet. The dorsal setæ, including those of the scutum, of one of the species without platelets varied considerably in length and stoutness. There were also specimens of *Leeuwenhoekia australiensis* (Hirst, 1925), *Guntheria bipygalis* (Gunther, 1939), and one specimen of the genus *Walchia*. Several specimens of *Guntheria bipygalis* had a single posterior plate, but were otherwise typical. This departure appears to be due to a developmental fault, as careful examination revealed occasional specimens with the single plate showing incipient division.

Of these species of Trombidiidæ, eleven, including *Trombicula deliensis* and *Neoschöngastia indica*, have not previously been reported from Australia. *Neoschöngastia perameles* Womersley 1939, *Neoschöngastia queenslandica* Womersley 1939, *Neoschöngastia smithi* Womersley 1939, and *Neoschöngastia derricki* were recently found in southern Queensland, but only the last species occurred in my collections.

Besides the larval trombids, there were several species of Lælaptidæ, specimens of *Listrophoroides expansus* Ferris 1932 (Listrophoridæ) previously taken from rats in the Marquesas Islands, and *Myobia ensifera* Poppe 1896 (Cheleytidæ).

Only occasionally was any other tick than *Hæmaphysalis humerosa* Warburton and Nuttal 1909 found on rats and bandicoots, and all of the exceptions appeared to be specimens of *Ixodes holocyclus* Neumann 1899. A few specimens of *Rhipicephalus sanguineus* Latreille 1806 were taken from a dog, and specimens of *Ixodes holocyclus* were found on man and dogs. An unidentified specimen of the genus *Aponomma* was found in the scrub near Cairns. I am indebted to Mr. D. C. Swan, of the Waite Agricultural Institute, Adelaide, and to Mr. D. J. W. Smith, of the Laboratory of Pathology and Microbiology, Brisbane, for assistance in identifying these ticks.

Polyplax spinulosa Burmeister 1809 was found on nearly all the rats and bandicoots, and a few specimens of *Læmopsylla cheopis* Rothschild 1903 were found on occasional animals.

Type specimens of the ticks and mites not previously reported from Queensland, with the exception of the specimen of *Walchia* which was accidentally destroyed, have been placed in the Waite Agricultural Institute and the South Australian Museum respectively.

Immunity.

I saw no cases of reinfection in humans. It is probable that the serum of a person once infected will subsequently have a non-specific rise and fall of agglutination titre. This apparently was what occurred in the case of Weil's disease cited above. To establish a definite second attack it is necessary to recover the rickettsia on each occasion.

Previously (Heaslip, 1940) I stated that an inapparent or non-fatal attack in mice gives immunity for at least two months. This is doubtful in some instances; it appears that a mild attack with a weak strain will not give protection against subsequent inoculation of a virulent strain. By means of the intraocular method in rabbits, cross-immunity was established between rat, bandicoot and human strains.

The facts that blood may be still infective for mice when Proteus OXK is agglutinated by the serum at high titre, and that agglutinins frequently do not appear until some time after convalescence is established, indicate that there is little if any direct relation between the production of agglutinins and resistance to infection.

Epidemiology.

Mathew (1938) has given an adequate description of the type of country where the disease occurs, and of the seasonal and occupational distribution of cases.

The patients listed in Table I lived in the coastal area between Cooktown and Ingham, the majority being resident in the Cairns district. It is probable, however, that the disease occurs elsewhere in northern Australia. What was probably an outbreak of tsutsugamushi was investigated by Wheatland (1924) near Sarina. No cases have been recorded from any place south of this. It is not known why the disease is restricted to the tropics in Australia. The rat reservoir is not limited geographically, nor are the larval Trombidiidæ which attack man and rats. However, it is probable that *Trombicula deliensis*, the suspected vector, is found only in the true tropics.

Although this mite constituted 90% of my collection and is found in many other parts of the tropics, it did not occur in the Brisbane collection. The latter was extensive and was taken from rats and bandicoots caught in the bush. Nor has *Trombicula deliensis* been reported previously from any other part of Australia. It is therefore possible that the distribution of the disease depends on the distribution of this mite.

It is obvious that the disease is partly occupational. For the rest it is mainly a matter of the proximity of dwellings to uncleared areas, and is therefore in a considerable measure preventible at relatively little cost. To clear, and keep cleared, the grass in the West Cairns area would not only destroy the mites, but would eliminate the food and shelter afforded to the rat and bandicoot population.

Comment.

As stated in the introduction, there seems to be no reason why the disease described in this paper should not be regarded as tsutsugamushi. Moreover, the observations recorded above support the conclusion of Lewthwaite and Savoor (1940). It is obvious that the disease in Queensland may show wide variations clinically, experimentally and serologically. There may be an eschar or no eschar, a rash or no rash, severe illness or mild illness; the blood may be highly infective or slightly infective for mice; and a high concentration of agglutinins may appear in the serum, or there may be little or none. The mortality rate at present is low, but in the cases which occurred near Sarina it was high, as it appears to have been amongst the early settlers on the Daintree River. Nevertheless, it is probable that only one strain of *Rickettsia* is responsible for all the cases, for the following reasons. (i) There appears to be no correlation between the various manifestations and either time or place of occurrence, and no grouping of the various manifestations. (ii) Although variations in virulence and pathogenicity may appear with different strains in certain passages in mice, ultimately, under uniform conditions, the same variations in duration of infection and in post-mortem findings will be observed with all strains. This has been tested with a large number of strains and by simultaneous observation of three strains—rat, bandicoot and human. (iii) Cross-infection tests in rabbits have shown the identity of three strains. (iv) With the exception of susceptibility of white mice, all the variations recorded from other countries have been observed in a circumscribed locality.

Finally, it seems probable that *Trombicula akamushi* (*Trombicula deliensis*) is the usual vector of the disease wherever it occurs.

Summary.

1. The results of the investigation of 54 human cases of tsutsugamushi in Queensland, Australia, have been presented and the reasons for the diagnosis have been given.

2. The possible vectors have been discussed and the reasons for suspecting the larval mite *Trombicula deliensis* have been presented. The ectoparasites found associated with the animal reservoirs are recorded.

3. Bandicoots (*Isoodon torosus*) and various species of rats have been infected, and found naturally infected, with tsutsugamushi.

4. Experiments with monkeys, rabbits, guinea-pigs and mice have been described.

5. White mice have proved highly susceptible to the disease and of great value in diagnosis. The importance of splenic enlargement has been demonstrated.

6. Serological findings have been recorded and the high degree of variation has been discussed.

7. A case of laboratory infection is described.

8. The fact that most humans who are affected are also infected with *Bacillus tropicus* is noted.

Acknowledgements.

In addition to those people mentioned previously in the text of this paper, I wish to express my sincere thanks to that goodly company the medical practitioners of Cairns, Dr. V. Byrne, Dr. F. Castellano, Dr. H. Flecker, Dr. T. G. Hewitt, Dr. A. Langan, Dr. E. Mansfield and Dr. D. Sword, and to Dr. S. Hambrett, medical officer in charge of the Commonwealth Health Laboratory, Cairns.

This paper is published with the permission of Sir Raphael Cilento, Director-General of Health and Medical Services, Queensland.

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Reviews.

BIOLOGICAL OXIDATIONS.

Oxidation is the fundamental chemical reaction of the living organism. This much Lavoisier realized; but he could not have guessed the devious routes and manifold stages by which the simple end products of oxidation are reached. In his monograph on biological oxidations, D. E. Green has reviewed the recent work on this complex subject, work to which he himself has made notable contributions. Information on the subject is still fragmentary, so that no connected account of it is yet possible. The author has therefore been obliged to present a series of chapters, with no very evident relation one to another, on the principal oxidations which have been studied in living organisms or in products immediately derived from them.

One common feature of these oxidations is that they do not occur spontaneously. The substrates or foodstuffs of living cells react with their appropriate oxidizing systems only in the presence of certain complex, specific, protein molecules known as enzymes. The author restricts his review to certain substrates and the enzymes which catalyse their oxidation under biological conditions. With the changes of energy which accompany these reactions, and which may be their *raison d'être* from the point of view of the living organism, he does not attempt to deal. For many of the reactions considered information on their energy exchanges does not yet exist.

The complexity of biological oxidation is further increased by the fact that very diverse oxidizing systems have been found not only in different organisms, but in different tissues of the same organism, and even in the same cells. It is tempting to imagine, in spite of the differences of detail encountered, that there may be some essential series of reactions common to all living things, or at least to the higher organisms. The nearest approach to anything of the kind is perhaps the cytochrome systems. In many different types of cell these serve to bring its substrates through the final stages of their path towards union with oxygen. But the author specifically warns his readers against the premature acceptance of such a generalization, and it must be remembered that with anaerobes the final oxidant is not oxygen.

One interesting feature to which this review draws attention is the number of instances in which vitamins or trace elements have been shown to be the essential parts of oxidation systems of living cells. They often act as coenzymes or prosthetic groups of the specific enzyme proteins. Among vitamins functioning in this way are thiamine (B_1), riboflavine (B_2) and nicotinic acid amide (PP factor), and among the elements (in simple organic combination), iron, copper and zinc. Certain avitaminoses and some other deficiency diseases may thus be due ultimately to disturbances of oxidation reactions in the cell.

The monograph will be found to be rather too specialized by the general medical reader. To him the chapter of greatest interest will probably be the introduction on the general properties of oxidizing enzymes. The book will be very useful indeed to the biochemist working on biological oxidation. The comprehensive series of recent references accompanying each chapter will be especially valuable to workers on the subject.

[1] "Mechanisms of Biological Oxidations", by D. E. Green; 1940. Cambridge: University Press. Melbourne: G. Jaboor. Demy 8vo, pp. 181, with diagrams. Price: 12s. 6d. net.

INFANT FEEDING.

In a brochure of thirty pages[1] Dr. Alan Moncrieff has reprinted the chapters on infant feeding from the eighth edition of "Lectures in Diseases of Children", by R. Hutchison and Alan Moncrieff. He has done this in response to requests from nurses, midwives, medical students and practitioners to whom he has lectured. He deals shortly and simply with the feeding of infants from birth to the age of three years. In the first section a short statement of general principles is made, and these are then applied to the management of breast feeding, feeding with cow's milk (liquid, dried and condensed), mixed feeding and the special feeding of the premature infant. In the preface the author states that he has "deliberately avoided any reference to units of energy in relation to the food values in the section on artificial feeding". He, however, feels it necessary to use some measurement, and he goes back to von Pirquet's "nem" or nutrition equivalent milk, which has never been widely accepted as a satisfactory measurement of food values. The following statement is made without any explanation, except that "the word equivalent is very loosely used and must not be misinterpreted; there is nothing to equal breast milk, and all bottle feeding is second best. One ounce of undiluted cow's milk is equivalent to one ounce of human milk. One and a half level teaspoonfuls of sugar are equivalent to one ounce of human milk. Three-quarters of a level teaspoonful of 'fat' is equivalent to one ounce of human milk".

It is difficult to understand why Dr. Moncrieff considers this an improvement on the generally accepted estimation of food values in Calories. It is impossible in a pamphlet such as this to give an adequate account of infant feeding; but for those who are interested in a short and clearly written statement of methods of infant feeding found satisfactory by an experienced pædiatrician, such as the author, this little book should prove useful.

MASSAGE AND EXERCISES.

The fourth edition of "Massage and Remedial Exercises", by Noel M. Tidy,[2] follows very closely the form of the third edition with two short new sections on *myositis ossificans* and the effects of gas poisoning.

A very large number of diseases is dealt with. The ætiology, pathology and general treatment are given in more detail than is usually found in works on this subject. Some of this would seem to be unnecessary. But the treatment as required to be given by the physiotherapist is well and fully described, and the exercises are set out in such a way as to be easily understood.

A special feature of this book is the excellent illustrations, all taken as moving pictures and reproducing the positions which give the best idea of each exercise.

Altogether this book can be very strongly recommended as a comprehensive contribution to the subject of physical therapy—a well-illustrated, well-written, authoritative book.

[1] "Infant Feeding", by A. Moncrieff, M.D., F.R.C.P.; 1940. London: Edward Arnold and Company Limited. Demy 8vo, pp. 34. Price: 1s. 6d. net.

[2] "Massage and Remedial Exercises in Medical and Surgical Conditions", by N. M. Tidy; Fourth Edition; 1939. Bristol: John Wright and Sons Limited. Demy 8vo, pp. 470, with illustrations. Price: 15s. net.

The Medical Journal of Australia

SATURDAY, MARCH 29, 1941.

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OF THE MEDICAL PRACTITIONER HIMSELF.

ONE who is in the habit of reading this and other medical journals has remarked that many articles appear in editorial columns and under the names of various authors on the medical practitioner, his relationships with and his duties to the general public, scientific problems, friendly society lodge practice, the war effort and so on, but that nothing appears to be written on the medical practitioner, what he is and what he owes to himself. The idea is stimulating, though it must be admitted at once that in the articles which our critic tells us have been published, a great deal about the medical practitioner himself will be found. However, it may be of interest and also perhaps of profit to think for a moment of how the medical practitioner sees himself or could see himself if his eyes were opened, of how other people see him and then of what he owes to himself.

Very few persons are able to see themselves as they really are, even if we except all that has been repressed and tucked away into the subconscious. When therefore we ask how the doctor sees himself, we ask in vain, for we shall never know anything much about anyone except ourselves; but we can use our imaginations. If then we imagine that each medical man could and was willing to reveal himself, what a patchwork we should see! It has been said that doctors in the mass are uninspiring; this is true enough, but the same can most likely be said of a similar group from any trade or profession. Let us then go further and for the moment embrace in our survey men of every group and station in life. In these circumstances none can doubt that a public display of minds would rival that of bodies for diversity and peculiarity; it would also provide surprises in plenty. How many minds, we may wonder, would there be set like a watchtower on a pinnacle of understanding so that the whole plain of life might be viewed from sunrise to sunset in one unbroken expanse? The answer "comparatively few" need occasion no surprise; understanding is seldom complete and the windows do not always stand widely open. Many a man who has a watch-tower to his dwelling finds the noonday glare too strong and either makes some of the windows fast with shutters or puts on blinkers. Later he is quite likely to forget that he has made any adjustment. There are many like this. They seldom look back in the direction of the sunrise, and if they do look towards the sunset, they do not think of the future as having much to do with the vista at their feet or with what is behind. But let us look at the medley of minds that we have imagined in display; let us suppose that we can look into them to estimate their size, inspect their contents and gauge their outlook on the world around. We shall find some so small that the contents are negligible. Others, a trifle larger, are set in such dull, flat and murky places that there is nothing much to see through any aperture that may be present. Other minds are of every size and of every shape and the furniture is as variable as both. Some are tidy and others a mere litter or jumble of foolishness and incoherence; and we shall find every grade of tidiness in almost every type of mind. But as we are thinking particularly of the mental outlook we shall look at the openings, the windows, that are to be found in each. Some have many; others few. The mind with many windows is fortunate, particularly if they are large. The mind with few or with a single window may be content, but contentment and joy in living will be increased with a realization that holes may be pushed in any of the walls to make the outlook wider. The mind of smug respectability will have long straight mullioned windows, rather heavily leaded and with only a small section near the top that can be pulled open by a cord of self-righteousness. In other minds the cobwebs have been allowed to gather through laziness and have partly obscured the vision. The generous mind has its casement flung wide and is "trellised with intertwining charities", it is readier to give than to receive and is of all minds the most to be admired. Some minds give out on the highway in many directions; others have a narrower prospect. In many we can take joy, some make us sad and others move us to pity. We are moved most of all by the mind, once roomy and maybe brilliant, but now so cramped that light comes through a mere chink in its walls while its windows have been boarded up in order to save trouble or because of some other deplorable human failing. To describe the many types of mental outlook is an impossible task, but the openness of a man's mind may be taken as a measure of the man himself.

We have imagined that doctors can see themselves as they really are and we have remarked that this is what few of us can do. Other folk are more discerning when they complain that doctors always talk "shop"; the truth is that many doctors find it hard to talk anything else and some, more is the pity, have few ideas on any other topic. Some of these dullards have begun life with first-rate mental equipment, but, instead of using it in a balanced fashion, have stuffed their minds with special learning until from habit or from a false idea of values they have become unwilling and eventually unable to climb out of the groove of their own making. The heroes and

heroines of the profession who make new year resolutions not to read anything but medicine and would even restrict their reading to their own particular branch of medical science, are heading for mental sterility and deserve all the pity and help that others can give them. Another danger which the medical mind has to face and of which it may be oblivious is that of self-adulation and conceit. The nature of a medical man's calling tends to engender these qualities—he is trusted by men in every walk of life and he ministers to them in the mysteries of birth, life and death; he may easily delude himself that he is something that he is not. This in the language of the hygienists is one of the special hazards of medicine. Closely allied to it is that of selfishness and intolerance which shows itself in the attitude adopted towards other members of the profession. The man who has nothing to fear from these tendencies or dangers is the man who can take a view of life as a whole, who lives not for today only but for tomorrow, who lives not to himself but for others. The watchtower mind of this man has already been described; it is a mind which in its highest development has a cosmic sense. This type of mind is not to be had for the asking. It is acquired by deliberate effort and self-sacrifice, by a widening of the interests and the sphere of activity, by a search for origins and meanings and by a refusal to entertain unworthy motives. The best way to set about the acquisition of an open and understanding mind is to prevent its closure, and from their experience in disease doctors know better than other men what preventive measures mean and what they can do. As an effort is made to acquire these attributes man will be able to see himself in increasing measure as he really is, and this is what he owes to himself. One of the surest signs of development in the right direction is a display of generosity of mind, and in the practice of medicine it will be seen in the relations between one practitioner and another. But it will be said that all this is idealism. So it is, but we must remember that the world stands in great need of idealists at the present time. We may not all attain the ideal—most of us are sure to fail—but the striving for it will make us better men and women and we shall by our efforts make life more tolerable for those around us.

Current Comment.

HUMAN INFERTILITY WITH SPECIAL REFERENCE TO THE MALE.

THE day is fortunately passing when the blame for a childless marriage was always attributed to the wife, for whom the sole therapeutic resort was dilatation and curettage of the uterus. Conception is a complex process in which the elaborate sexual mechanisms of both male and female must function efficiently and harmoniously. There are a multitude of reasons which may account for the infertility of any couple, but investigation and interpretation of observations have progressed to a point at which little of a factual nature needs to remain obscure in their physical status, excepting only the question of whether or not mature ovulation occurs. A recent symposium[1] describes the methods for investigating the problem, with particular reference to the male partner. S. R. Meaker and S. N. Vose, discussing the nature of human infertility, point out that in only 30% of cases is there a single factor which alone and by itself renders conception impossible. In the majority of cases there is a multiplicity of factors, usually each of lesser degree, and often involving both husband and wife. One or two of these Nature would overcome, but when several of them act together the combined fertility of the couple is reduced to a level at which reproduction becomes impossible. These minor faults of course often accompany the less common major ones. A survey of a large series of cases at the clinic of these workers revealed that the average number of factors contributing to the infertility of each couple was 4·8, and in 90% some fault was found in both members of the pair. It is obvious therefore that the study of the childless couple must be thorough and can with advantage follow a routine which does not stop when a single cause of sterility is found, but proceeds until the examination is complete. It must include a general physical examination for evidence of systemic disease. Focal sepsis is important for it depresses spermatogenesis. Evidence of endocrine disease, especially hypothyreoidism, hypopituitarism and testicular atrophy, must be sought. The family history and sex life of the couple must be known, for fertility runs in families and excess reduces potency. The gynæcologist must determine the state of the vagina, of all parts of the uterus and of the ovaries and the patency of the Fallopian tubes. Finally a specialized investigation must be made of the genitalia of the husband. It is to this aspect of the problem that the remainder of the symposium directs attention.

The diagnosis and treatment of sterility in the male are discussed by H. A. R. Kreutzmann. Special questions to be asked of the patient refer to the incidence and complications of gonorrhœa, syphilis and mumps, and genito-urinary operations or accidents. The examination of the genitalia should start with the penis, which may show scars, hypospadias or meatal strictures. The urethra must be examined for stricture or discharge. Rectal examination must determine the size and consistency of the prostate and seminal vesicles, for a chronic inflammation of either can contribute to infertility. Each testis must be examined for cryptorchidism, atrophy, nodules on the epididymis or thickening of the *vas deferens*. The seminal fluid must be carefully examined, and the number of sperms per cubic millimetre, the percentage of abnormal forms and the degree of motility of the sperms determined. It is desirable that the sperm count of the semen should be at least several million per cubic millimetre, but it may exceed this by a great deal. If sperms are completely absent, the cause may be an inflammatory obstruction somewhere between the *globus major* of the epididymis and the ejaculatory ducts, or it may be agenesis or atrophy of the seminiferous tubules of the testis. Reduction in the number of sperms, increase in abnormal forms or decrease in their motility may be caused by focal or local sepsis, partial seminal obstruction or depression of the activity of the seminiferous tubules. A still more specialized examination is therefore necessary for the full elucidation of the problem. If the ejaculatory ducts are stenosed the patient gives a history of a burning sensation in the perineum at the moment of ejaculation, and the seminal vesicles fail to empty on massage. The patency of the *vas deferens* may be determined by exposure of the vas through a small incision in the scrotum and injection of a coloured fluid through it. It is wise to use a different colour on each side. A solution of mercurochrome on one side and of a protein silver preparation on the other have been found serviceable. The real state of the testis may be determined by a simple biopsy, described in detail by C. W. Charny. The skin of the scrotum is stretched tightly over the testis, and under local anæsthesia a small incision is made through layer after layer until the glistening visceral layer of the *tunica vaginalis* is reached. This and the underlying *tunica albuginea* are nicked, and gentle pressure is applied to the testis to force through the nick a small bead of testicular tissue. This is cut off with fine

[1] *The Journal of the American Medical Association*, October 26, 1940.

scissors. It is preserved in Bouin's solution, mounted in paraffin for section and stained with hæmatoxylin-eosin.

When this complete examination is made, and only then, can the doctor fully appreciate the problem he is handling in any infertile couple, and only with this full understanding can he undertake the most rational treatment and give the most accurate prognosis. Obstructive lesions can sometimes be remedied by probing the ejaculatory ducts through a urethroscope and the vas through the scrotal incision. Focal or local sepsis should be treated. Chronic seminal vesiculitis responds to prolonged heat and massage. A moderate degree of hypospadias can be treated by operation or more easily by the wearing of a condom with a small hole in the end during intercourse. For cryptorchidism operation or glandular therapy can be tried, but after puberty the spermatogenic function of the testis is not often recovered. The infertility of hypothyreoidism responds well to thyreoid medication. If oligospermia, aspermia or a high proportion of abnormal forms is due to a primary hypofunction of the testis, therapy with one of the androgenic substances is of value. The most often used of these is testosterone propionate. Gonadogens and œstrogens are also sometimes used with benefit. The results vary widely in different cases, even though in all the fault seems to be a primary testicular hypoplasia. Some men react with a large increase in the number of active sperms, in some there is no change and in some there is even a decrease in the number of normal forms. For this reason glandular therapy must be periodically checked by semen examinations or even by testicular biopsies. The practice of glandular therapy for sterility without regular and careful control must be strongly condemned. So too should we condemn the attempt to manage the problem of human infertility without the thorough examination outlined above. Obviously it is not the task of the general practitioner, but calls for the service of a group which includes a urologist, gynæcologist and general physician. The challenge of a falling birth-rate and of the discontent that arises from this problem should be sufficient to lead to the adoption of measures that will at least enable it to be handled as efficiently as possible.

THE DIAGNOSIS OF EXTRAUTERINE PREGNANCY.

In a speciai article on the diagnosis of extrauterine pregnancy that he wrote for this journal in 1931, E. R. White, of Melbourne, stated that if ectopic pregnancy was thought of it would not often be overlooked. This is, of course, true, but for all that most general practitioners and many gynæcologists can recall cases in which diagnosis has been far from easy even when extrauterine pregnancy has been considered as a possibility from the inception of the patient's illness. One of the most salutary ways in which a diagnostic lesson can be learned is by a study of wrong diagnoses, especially if the errors are those of the person who is learning the lesson. Clinicians do not often publish their errors, but when they do, the opportunity of discovering why the errors were made should not be lost. In a recent clinical study of forty consecutive cases of extrauterine pregnancy, R. L. Dodds admits to having opened six abdomens on his own wrong diagnosis of extrauterine pregnancy; he also refers to mistakes made by other clinicians who saw some of the patients in the same series, and discusses the difficulties.[1] In four of the cases in which Dodds was responsible for the error acute salpingitis was found when the abdomen was opened; in one instance there was a threatened miscarriage and in the last case all the abdominal viscera were normal, and it was not until two days later that vesicles typical of *herpes zoster* appeared. Of the forty patients thirty-six came under Dodds's care with some sort of diagnosis and these included four who had been sent on from other hospitals. The diagnosis was correct in only seven of these thirty-six cases. In twelve of the twenty-nine cases in which the diagnosis was incorrect, no mention was made of any part of the genital tract. This is most surprising. In five of these twelve cases the diagnosis was "abdominal pain", in three it was appendicitis, in one case the diagnosis was perforated gastric ulcer, in one it was acute intestinal obstruction, in one food poisoning and in one internal hæmorrhage. Of the remaining seventeen cases seven were labelled miscarriage and ten were thought to be other conditions of the genital organs such as menorrhagia or salpingitis.

The signs and symptoms of extrauterine pregnancy are generally described as including amenorrhœa, pain, bleeding from the uterus, the presence of a swelling in the pelvis and possibly an enlargement of the uterine body. Dodds makes some interesting observations on some of these. In thirty-seven of his forty cases the menstrual history was noted and in fifteen of these amenorrhœa "could not be established as a sign". In one of these fifteen cases there was no time for menstrual irregularity to occur, for the tube ruptured and the patient died within three weeks of the last menstrual period. In the other fourteen cases the menstrual period occurred at the expected time, and in only two of the fourteen was a correct diagnosis made before the patient's admission to hospital. The wrong diagnoses were so wide of the mark that no possibility of any kind of pregnancy had been considered, not even miscarriage. In spite of the absence of amenorrhœa it is important to note that every one of the fourteen patients had bleeding which was excessive either in quantity or in duration or both; Dodds insists, as other observers have done, that this menorrhagia is important from the diagnostic point of view when amenorrhœa is not present. "Outside" diagnoses were made in twenty cases in which amenorrhœa had occurred; in more than half of these pregnancy was considered, for in five a correct diagnosis of ectopic pregnancy was made and in seven the diagnosis was a miscarriage. On his figures, Dodds is more than justified in his statement that while amenorrhœa is a sign of great help, extrauterine pregnancy must sometimes be diagnosed when it has not occurred. It is interesting to note that the acuteness of the condition in Dodds's cases was not an aid to diagnosis, as most practitioners would expect it to be—in nineteen acute cases three correct diagnoses were made and in twenty-one subacute cases four. In thirteen of Dodds's cases pain was trivial or absent and he finds that abdominal pain is of small value in the differential diagnosis. Unlike Bethel Solomons, he attaches considerable importance to shoulder pain which was present in seven of his nineteen acute cases. He goes so far as to state that shoulder pain in a woman suffering from an acute abdominal condition is pathognomonic of extrauterine pregnancy.

In the differential diagnosis the conditions most likely to cause difficulty are miscarriage and acute salpingitis. Dodds lays particular emphasis on the difficulty associated with acute salpingitis, "for here the treatment is not surgical". He points out that if diagnosis is not possible at once, it will emerge as a result of the failure of conservative treatment. He uses the following sentence which, for lack of an Irish term, we shall call a *bon mot*: "An acute salpingitis which gets worse in spite of four day's efficient medical treatment, is likely to be an ectopic pregnancy." The point is important, and it is well to recall White's statement about a procedure that should be used when occasion demands it: "It may be necessary to open into the posterior fornix, when dark blood and clots will escape to establish the presence of an extrauterine gestation." This is a reminder that extrauterine pregnancy may come under the notice of the medical practitioner in either an acute or a subacute form. As a final indication of the difficulty in diagnosis that may be experienced by some clinicians, it will be useful to point out that in nineteen acute cases there were three "outside" correct diagnoses and in twenty-one subacute cases four. This, too, in spite of the fact that in ten of the acute cases the patients had unmistakable signs of hæmorrhage.

[1] *Proceedings of the Royal Society of Medicine*, October, 1940.

Abstracts from Medical Literature.

PÆDIATRICS.

Sulphapyridine in Pneumonia of Infancy and Childhood.

JEROME S. LEOPOLD AND IRWIN PHILIP SOBEL (*Archives of Pediatrics*, November, 1940) have reported a second season's experience with sulphapyridine in 56 consecutive cases of pneumonia in children. Blood counts and examination of the urine were carried out every day while the patients were receiving the drug and every other day for at least four days thereafter. A specimen of blood for incubation and a naso-pharyngeal or pharyngeal swabbing for incubation for pneumococcus typing were taken in each case before the therapy was instituted. In 45 cases one or more determinations of the sulphapyridine content of the blood were performed. In every case pneumonia was demonstrated by X-ray examination. The age distribution was from two months to eight years. Thirty-seven of the patients were three years or younger, 25 two years or younger, and 15 were a year or younger. The authors were of opinion that the pneumonia was somewhat more severe than in the previous year. The dosage of sulphapyridine was the same as that previously used, with the exception that double the amount was frequently given in the first and occasionally the second dose. Six patients received 10% sulphapyridine in glucose. The total 24-hour dosage of sulphapyridine as glucose sulphapyridine was divided into three doses and administered every eight hours. No apparent relationship existed between the blood sulphapyridine content and the therapeutic efficacy of the drug. In 38 of the 56 cases the temperature dropped to normal within thirty-six hours or less after the administration of the drug was begun. In 31 this result was obtained in twenty-four hours or less. The authors conclude that their second year's experience with sulphapyridine in the treatment of pneumonia in infancy and childhood has been as satisfactory as the first. There were no fatalities in the second series, in spite of the fact that 45% of these patients were infants of two years of age or younger. Oxygen, antipneumococcus serum, blood transfusions, infusions, expert nursing and non-specific measures must be used as indicated in addition to chemotherapy in order to bring the mortality from pneumonia to its lowest possible level. With the exception of occasional vomiting, no toxic manifestations of chemotherapy were detected in the authors' series. Although examination of the urine and complete blood counts were frequently done in each case, not a single instance of granulopenia or microscopic hæmaturia could be detected. The authors believe that two factors contributed to the absence of harmful effects of the drug. The first was an abundant fluid intake. The daily amount by mouth during the administration of the drug averaged 50 cubic centimetres per pound of body weight per patient. This was liberally supplemented whenever necessary by transfusions and intravenous infusions of 5% glucose solution. The second factor was the length of time during which sulphapyridine was administered. A divergence of opinion exists in this regard, but the authors think that it is best to avoid prolongation of the administration of sulphapyridine whenever possible. They consider that in general the schedule which most closely approximates the ideal consists in the continuance of sulphapyridine therapy for one or two days of normal temperature followed by the abrupt termination of administration of the drug.

Treatment of Pneumonia in Children with a Single Dose of Sulphapyridine.

LOUIS PLATT (*American Journal of Diseases of Children*, November, 1940) comments on the fact that although the effectiveness of sulphapyridine in the treatment of pneumococcal infections has been accepted generally, the problem of determining the best method of administration still remains. The most commonly accepted daily dosage of the drug is approximately 0·2 gramme per kilogram of body weight in divided doses and given for from two to four days. A level of four milligrammes of free sulphapyridine per hundred cubic centimetres of blood has been accepted as satisfactory and favourable therapeutic results have been obtained with levels as low as two milligrammes per hundred cubic centimetres. The author decided to determine the levels in the blood and to evaluate clinical results following the administration of one large dose of sulphapyridine to see whether effective levels in the blood, maintained long enough to produce a cure, would result. In the series studied there were 41 patients, ranging in age from two months to twelve years. Six children were less than six months old, and a total of 15 were under one year of age. The amount of sulphapyridine given was 0·3 gramme per kilogram of body weight (two grains per pound) as a suspension in either milk or a formula. Although in a few instances considerable coaxing was necessary before the children took this large dose, in no case was it necessary to resort to gavage. After administration of the drug nothing was given by mouth for four hours. In three instances immediate vomiting necessitated another dose. After four hours the children were given food as desired and fluids were forced. In the cases included in the study this single dose method gave satisfactory levels in the blood in most instances and the clinical results were as good as, if not better than, those in the group that received multiple doses. If the results are as good, it would seem desirable to adopt a single dose method because it is simpler, interferes less with the child's rest and sleep at a time when he is most ill, demands less of the nursing staff and decreases the incidence of the most common toxic effect, nausea and vomiting. For the ordinary attack of uncomplicated pneumonia it would appear that 0·3 gramme of sulphapyridine per kilogram of body weight administered in one dose will give satisfactory results. Lower doses are not so consistent in giving levels in the blood above four milligrammes per hundred cubic centimetres. Although the lower levels gave satisfactory results, it would seem safer to use the larger dose. If it is thought desirable to maintain levels in the blood above four milligrammes per hundred cubic centimetres for more than twenty-four hours, the dose can be repeated at eighteen to twenty-four hour intervals. Such repetition is probably advisable when the patient cannot be watched closely, and would probably decrease the number of cases in which a secondary rise in temperature occurs.

Significance of the Tonsils in the Development of the Child.

ALBERT D. KAISER (*The Journal of the American Medical Association*, October 5, 1940) reports that follow-up studies of children whose tonsils had been removed and of those whose tonsils had not been removed were undertaken to show the trend of certain complaints over a ten-year period in the two groups and to note the effect of the tonsils on the physical development of the child. It was shown that considerably hypertrophied tonsils and tonsils that are repeatedly inflamed, giving rise to attacks of tonsillitis and cervical adenitis, frequently do impair normal physical development. When such a condition exists after the age of four years, it is advisable to have the tonsils removed, and it may be expected that at least 50% of the children so treated will be materially improved. Such infections as the common cold, *otitis media*, sinusitis and laryngitis may unfavourably influence the child's normal development. It cannot be demonstrated that the tonsils are often a causative factor in these infections; consequently tonsillectomy does not offer a solution for their eradication, save in exceptional cases. It could not be shown that the incidence of such diseases as bronchitis, pneumonia and tuberculosis was reduced after tonsillectomy. The tonsils are not often responsible for pulmonary infections. Rheumatic disease and nephritis constitute serious handicaps to a child's normal development. Recent studies show that the tonsils play a less significant role in the causation and treatment of these diseases than was formerly supposed. However, a reduction in the number of throat infections following tonsillectomy has a beneficial effect in the rheumatic subject. There has been a significant decline in the mortality and morbidity rate of children during the last three decades. There has also been an improvement in the physical development of children in America. The application of the new discoveries in the field of nutrition, immunology and sanitation has contributed much to this favourable situation. When proper consideration is given to these factors, it is evident that the tonsils are not so great a menace to a child as has been frequently suggested. There is substantial evidence that in about 20% of the children the tonsils are either hypertrophied or diseased and therefore have an unfavourable influence on the physical development of the child. Such tonsils should be removed.

ORTHOPÆDIC SURGERY.

Chemotherapy and Compound Fractures.

ROBERT W. JOHNSON (*The American Journal of Surgery*, June, 1940) recommends the routine use of sulphanilamide in all serious compound fractures. The drug is used as a prophylactic for the prevention of streptococcal and gas bacillus infection. There is no omission or modification of thorough surgical toilet of the wound or of *débridement* of necrotic tissue. Early and adequate reduction and splinting of the fracture

are important, and antitetanus serum is to be given. A large initial dose of 60 to 80 grains of sulphanilamide is administered, with subsequent four-hourly doses of 10 to 20 grains, continued for a period of three to ten days. A blood concentration of five to eight milligrains *per centum* is sufficient for prophylaxis. A total of thirty-six patients treated in this way are reviewed by the author. In only four did serious infection occur, and in these the use of the drug had been commenced too late for effective prophylaxis.

Ruptured Quadriceps Tendon.

FRANCIS M. CONWAY (*The American Journal of Surgery*, October, 1940) reports cases of rupture of the quadriceps tendon. Reviewing the history of treatment of this injury, the writer points out that hot packs, poultices and splinting were the only forms of treatment applied until 1878, when Lister practised suture of the tendon under antisepsis. Records show that the majority of cases occur in men between the ages of thirty and fifty years. Animal experiments appear to prove that severe strain on a normal muscle does not cause rupture of the tendon, but rather the muscle will tear away from its origin or insertion, or rupture across the muscle belly or at the musculo-tendinous junction. Also investigation reveals that efficient blood supply to the tendon is of more importance than the presence of the tendon sheath. These observations strengthen the belief that predisposing causes may be present. Examples of these are: tuberculous tenosynovitis, gonococcal tenovaginitis, typhoid or *lues*. These factors must be considered in the so-called "spontaneous rupture" following slight trauma. The type of injury causing rupture by indirect violence is usually due to vigorous muscle contraction in any effort to avoid an impending fall. The inferior patellar margin and the superior patellar insertion are the most common sites. The patellar tendon is divided into three layers: a superficial layer arising from the *rectus femoris*, a middle layer from the medial and lateral vasti, and a deep layer from the *vastus intermedius* (or crureus). The lastmentioned lies in close proximity to the suprapatellar synovial pouch, and involvement of this portion of the tendon is responsible for the hæmarthrosis commonly found in quadriceps tendon rupture. The trilogy of symptoms of rupture in recent cases is pain, crepitus and loss of function. Pain is violent and immediate, crepitus is not a constant accompaniment and, when present, will be confounded with crepitus due to fracture. The function loss is found in inability to extend the knee. In cases of rupture above the patella examination reveals absence of fullness due to the suprapatellar pouch. In infrapatellar rupture the classical feature is upward displacement of the patella. Hæmarthrosis indicates an extensive injury. X-ray examination will demonstrate any associated bony damage. In long-standing cases there is inability to extend the knee fully or to walk up an incline. Quadriceps atrophy will be present. Treatment in all cases aims at an accurate apposition of the ruptured parts and the prevention of quadriceps muscle atrophy. In cases of partial rupture when full function is present, operation is not essential, but suture will obtain a more certain and complete end result. In suprapatellar rupture the immediate concern is to determine whether or not the knee joint has been ruptured. Synovial closure is the first step in such cases, followed by suture of the tendon in layers. When the rupture is close to the upper pole of the patella, fascial suture *plus* fixation to the body of the patella may be necessary. In infrapatellar rupture involving the patellar rim, fascial or heavy catgut fixation *plus* drilling through the patellar substance is the most satisfactory procedure. Avulsion of the most inferior portion, with or without avulsion of the bone, demands fixation to the tibia. Repair in long-standing cases, to be successful, depends on four main factors: (*a*) ablation of all old scar tissue, (*b*) restoration of tendon continuity, (*c*) rehabilitation of the quadriceps muscle, and (*d*) absence of intraarticular complications.

Fractures of the Head and Neck of the Radius.

R. C. MURRAY (*The British Journal of Surgery*, July, 1940) reviews 459 closed fractures of the head and neck of the radius. These constitute 44% of all elbow fractures treated at Liverpool Royal Infirmary. This type of fracture is the commonest about the elbow joint. Fractures of the radial head are classified into five groups: from a simple crack to the completely crushed and distorted comminuted fracture. A rare type called a "flake fracture", which is distinguished by a small chip of bone from the orbicular margin, is included. Fracture of the neck is commonly a greenstick fracture in children, but does occur in adults. In regard to the type of injury, the author believes that the commonest cause is a fall on the forearm, resulting in abduction together with flexion of the elbow. He disagrees with Cutler and other authors, who hold that direct violence is more commonly responsible. Tearing of the medial ligament of the elbow joint, occasional associated fracture of the lateral epicondyle, and operative proof of damage to the capitellum or its articular cartilage, strengthen the former view. Eighty-seven *per centum* of the patients were treated conservatively and 13% by operation. Conservative treatment consisted of resting the elbow in full flexion for ten days to three weeks, according to the severity of the lesion. In crack fractures without displacement active movements were allowed from the start. Patients with more serious fractures were permitted active movement after three weeks. Wrist and shoulder movement was insisted on in all cases from the beginning. Massage and passive movements were abandoned as harmful, after use in a few cases. Manipulation was found useful in displaced fractures of the neck. Open operation was performed in cases of gross comminution, marginal fractures with marked depression of a fragment, loose fragments in the joint, and fractures of the neck in which manipulation had failed to correct the angulation. Three types of operation were used: (*a*) total excision of the head, (*b*) partial excision, (*c*) open reduction for fracture of the neck. The results were assessed according to the range of movement and the function obtained. Of the group in which conservative treatment was adopted, only 37% of the displaced marginal fracture type resulted in full range of movement: but painless and strong function was obtained in 56% of cases, and in a further 44% the patients had only minor symptoms. Limitation of extension occurred in 92% of all cases, limitation of flexion in 31%, and of pronation and supination in 23%. The author draws attention to the fact that even 30° to 40° of limitation of movement seldom affects the usefulness of the elbow. Reviewing the cases in which operation was performed, the author states that the earlier the operation, the better will be the result. Many patients in this group had considerable limitation of movement, but function was strong and painless. Total excision of the head resulted in better range of movement than partial excision, and it was found that good results from partial excision were obtained only in cases in which simply a loose fragment was removed. In those cases of fracture of the neck in which open reduction was practised, the results were not so good as those treated by manipulation. The author believes this is due to the formation of adhesions between the neck and the orbicular ligament, which usually required at least partial division.

Hammer Toe.

A NEW procedure for correction of hammer toe is described by Seth Selig (*Surgery, Gynecology and Obstetrics*, January, 1941). The most generally used procedure is a wedge resection of the proximal interphalangeal joint. Following this operation, splinting for six weeks is usually required before bony union is obtained. In the past it has been difficult to arrange sufficient immobilization of the phalanges to permit of ambulatory treatment. The author's operation consists of passing Kirschner wire through the three phalanges of the affected toe. The joint is exposed by a longitudinal or transverse incision in the usual manner. The distal end of the first phalanx and the proximal end of the middle phalanx are then denuded of cartilage and accurate apposition is obtained to correct the deformity. Any bone to be removed should preferably be taken from the first phalanx. If any flexion deformity is present in the terminal interphalangeal joint, this is corrected by manual stretching. The Kirschner wire, forty-five one-thousandths of an inch thick, is then inserted by means of a hand chuck, through the tip of the toe, across the terminal interphalangeal joint and second phalanx, to appear at its denuded proximal end. Correct apposition of the phalanx at the site of operation is next obtained, and the wire is continued from the distal end of the proximal phalanx, along the shaft of this bone, for three-quarters of its length. The average length of the first phalanx in the adult second toe is between one and one and a quarter inches. In the case of other toes, the length must be determined by X-ray examination. To avoid subsequent wandering of the wire, the end protruding at the tip of the toe is bent. The wound is closed after it has been determined that there is no movement between the phalanges. Gauze soaked in collodion is now applied round the toe and held firm with strapping. The patient is allowed to walk immediately with a cut-out shoe. Stitches and wire are removed after six weeks. The results are satisfactory. Movement at the terminal joint returns after the removal of the wire. Infection did not occur in the writer's series of twenty toes operated on in this manner.

British Medical Association News.

SCIENTIFIC.

A MEETING of the Victorian Branch of the British Medical Association was held on February 5, 1941, at the Medical Society Hall, East Melbourne, DR. H. BOYD GRAHAM, D.S.O., M.C., the Acting President, in the chair.

Two Common Tropical Diseases.

DR. P. A. MAPLESTONE, research helminthologist at the School of Tropical Medicine, Calcutta, read a paper entitled "Two Common Tropical Diseases" (see page 377).

DR. H. BOYD GRAHAM, from the chair, expressed on behalf of those present appreciation of the eminently practical and masterly way in which Dr. Maplestone had referred to the appropriate treatment of malaria and amœbic dysentery. He mentioned that it was the custom not to allow formal votes of thanks at the monthly meeting, but asked Dr. Maplestone to take the thanks for granted. Dr. Graham then welcomed certain visitors who were present, and invited Professor English, professor of gynæcology and obstetrics at the King Edward VII College of Medicine, Singapore, to address the meeting, and said that Dr. Maplestone would be prepared to answer questions on the matters he had touched upon in his address.

PROFESSOR J. S. ENGLISH said that it was a privilege to be at the meeting and to have heard so informative an address; the Calcutta school was regarded in Malaya as the authority on malaria and helminthiasis. Helminthiasis complicated pregnancy at Singapore much more frequently than did malaria, though he thought he could say that the malarial complication occurred about two hundred times annually. A point of interest was the effect of quinine on pregnancy; Professor English had observed that the effect was negligible and that the fact of pregnancy was not a contraindication to its use; even if the malaria was not cured, the pregnancy was usually well tolerated. The worm infestations caused more debility than any of the infections. In his early days at Singapore a grave prognosis was indicated in the resultant anæmia when the hæmoglobin percentage fell below 25 and the number of red cells was less than 1,000,000 per cubic millimetre; such a state would be regarded as serious but by no means hopeless nowadays.

DR. KENNETH SMITH emphasized the importance of the warning given by Dr. Maplestone at the end of his paper, not to forget the common diseases as likely causes of symptoms in returned soldiers that there would be a tendency to ascribe to tropical diseases. He asked Dr. Maplestone to express an opinion as to the maximum length of time after a person had left a malarial area within which credence could be given to reports that malarial organisms had been found in blood preparations. Dr. Smith mentioned that the latest authentic record of the Repatriation Commission was that organisms had been found in 1925 when the disease had been contracted not later than 1919. He also asked Dr. Maplestone whether injections of adrenaline were considered to be of value in forcing the parasites into the blood.

DR. FRANK TRINCA asked four questions: (i) whether in chronic malaria excessive quinine prophylaxis was likely to bring about a poor response to treatment with quinine; (ii) whether Dr. Maplestone held the view that arsenical preparations exerted a synergistic toxic effect on the organisms; (iii) whether, as he had heard from a medical correspondent in Algiers, "Kaomagma" was most effective in the treatment of intermittent dysentery arising out of the last war; and (iv) whether, in view of Dr. Maplestone's statement that quinine was rapidly eliminated from the blood stream, it would not be preferable to use quinine in oil.

DR. CURZON referred to the occurrence in Europe of amœbic abscesses in the upper lobes of the lungs as well as in the liver; he mentioned that in pre-emetine days the mortality of hepatic abscess was approximately 90%, but that it had been reduced to about 10% by means of emetine treatment. He asked Dr. Maplestone to give more information about the occurrence of abscesses elsewhere than in the liver.

DR. SALTER asked for information about the time to cease the administration of quinine which was being given in the treatment of a malarial attack.

Dr. Maplestone, in reply to Dr. Kenneth Smith, said that organisms might be found in the blood after an attack of malaria for two or three years, but rarely after that time. The outside limit was approximately seven years. With reference to the use of adrenaline for obtaining positive blood findings, he knew that it had been recommended a great deal, and though of value it was not infallible.

In reply to Dr. Trinca, Dr. Maplestone said: (i) He had gathered that Dr. Trinca's experience of malaria had been gained in the Jordan Valley during the last war. He was dealing with a highly sensitive population, among whom transmission would be rapid and the disease much more acute than was ever the case in Calcutta. In those special circumstances the impression might have been gained that quinine prophylaxis in excessive amount might interfere with treatment. While he recognized that analogies were dangerous, he thought it might be of interest to refer to the fact that workers in trypanosomiasis had obtained arsenic-fast strains in animals. (ii) While arsenic was of undoubted value in the treatment of the associated anæmia, in which there was no loss of iron from the body, there was no doubt that it was not so good as quinine in malaria. (iii) Dr. Maplestone was unable to express an opinion about the value of "Kaomagma" in dysentery, but warned Dr. Trinca that probably other foreign correspondents would advocate the use of some one or other of the numerous drugs available; statistically, there was no definite cure. (iv) Quinine introduced intramuscularly did not disappear as it did by the intravenous route; but Dr. Maplestone could not advise the intravenous use of quinine in oil. In fact it was not necessary to inject quinine at all unless the patient was unconscious or could not retain it when given it by mouth; the necrotic abscesses were very troublesome. In reply to Dr. Curzon, Dr. Maplestone said that amœbic abscesses were likely to form in the bladder, in the lungs and almost anywhere, including the skin itself; but wherever they formed they reacted to emetine. In reply to Dr. Salter, Dr. Maplestone said that quinine treatment should be discontinued within three weeks and should not be used again unless the patient relapsed; if no relapse occurred, it could be taken that the patient was cured.

Medical Societies.

THE MEDICO-LEGAL SOCIETY OF VICTORIA.

THE Medico-Legal Society of Victoria has arranged the following programme for 1941:

Saturday, March 29.—Speaker: Mr. R. Vroland. Subject: "Our Professions and the Future."

Saturday, May 17.—Speaker: Professor G. W. Paton. Subject: "The Development of Forensic Medicine."

Saturday, September 27.—Speaker: Dr. Ralph McMeekin. Subject: "Functional Disorders and Workers' Compensation Legislation."

Saturday, November 15.—Speaker: Dr. Clive Fitts. Subject: "Pride and Prejudice."

The annual dinner will be held on Saturday, July 19, 1941, and particulars in connexion with that function will be communicated to members in due course.

The meetings are to be held at the Medical Society Hall, Albert Street, East Melbourne, and will commence at 8.30 o'clock p.m.

Medical practitioners who wish to join the society are invited to communicate with Dr. R. J. Wright-Smith at the Royal Melbourne Hospital.

Naval, Military and Air Force.

WIRELESS TRANSMITTING APPARATUS (POSSESSION) ORDER.

IN the issues of March 1 and 8, 1941, the attention of readers was directed to an order to come into force on April 1, 1941, and dealing with the control of diathermy and other high frequency apparatus. This order has been made under the National Security (General) Regulations and requires that no person, association, company, hospital, clinic or institution shall possess or operate the following types of apparatus without a licence, if such apparatus develops power exceeding 10 watts at frequency exceeding 10,000 cycles per second:

(*a*) Diathermy or other electro-medical apparatus in which valves or spark coils are used, not including normal modern types of violet ray, ultra-violet ray, infra-red ray, or X-ray apparatus or medical shocking coils.
(*b*) High frequency furnaces.
(*c*) Eddy current heating apparatus.
(*d*) High frequency testing oscillators.

The Postmaster-General's Department announces that applications for licence must be made on a special form which may be obtained from the Senior Radio Inspector in each State.

Before a licence to operate any of the above-mentioned equipment is issued, the room or premises in which the apparatus is installed must be suitably screened. The Postmaster-General's Department has issued the following specifications for the screening of rooms or cubicles.

Methods of Screening a Room or Cubicle.

Introduction.

This instruction is issued for the information of architects, builders, hospital authorities *et cetera*, who may be interested in the question of screening as a method of suppressing radio interference.

The methods of screening described are capable of suppressing interference to a very low level on long, medium, short and ultra-short waves, and are usually found to be satisfactory for the suppression of interference with radio reception, although in some extreme cases it may be found necessary to adopt more elaborate measures.

If circumstances warrant such a course, supplementary information will be published by the department. It would therefore be advisable for those interested to ascertain from the senior radio inspector if any further particulars are available.

General.

1. *Screening Materials Available.*—A number of materials are available for screening purposes, including sheet metal (such as aluminium, zinc, copper, or galvanized iron), metal foil supported on a paper backing and sometimes described as "metallized paper", and wire gauze. Half-inch wire netting is less satisfactory; when it is used care must be taken to see that all the cross-overs in the netting are well soldered. Paints having a metallic base are useless for screening purposes, as the binder insulates the particles of metal from each other.

2. *Relative Costs of the Various Screening Materials.*—It is not practicable to quote actual costs of screening a room, owing to day-to-day variations in the market prices of metals and the widely varying local charges for labour on a specialized work of this nature. An architect or a firm of builders or decorators will probably quote a definite price if informed of the method of screening proposed and given full particulars of the room(s) or cubicle(s) concerned.

3. As a general rule it may be said that the two cheapest satisfactory screening materials are metallized paper and wire gauze. For application to an existing room, metallized paper is generally preferred, as it may be applied to the walls and ceiling in a similar manner to wall-paper. In the case of a proposed building, wire gauze is, however, to be recommended. It has the advantage that it can be attached to the walls, ceiling and floor before they are plastered or otherwise finished. Not only is the gauze hidden from sight, but it will last longer than metallized paper.

Screening an Existing Room.

4. *Walls, Ceiling and Doors* (see Figures I and II).—The metallized paper advocated for this purpose is known in the trade as "paper-backed aluminium foil"; it consists of aluminium foil approximately 0·005 in. thick with a backing of thin paper for stiffening purposes. It is desirable that there should be as few joints as possible in the screen, and the metallized paper should therefore only be obtained from a firm which is able to supply it in reasonably large pieces. Several firms supply the paper in rolls of considerable length and having a width of 20 inches. The edges do not require trimming.

5. The paper should be applied to the walls and ceiling in the same way as ordinary wall-paper, with the metallized surface facing *inwards towards the room.* Any paperhanger's paste may be used, provided it does not contain strong alkali, as this attacks the aluminium surface. Flour and water paste with added size is also suitable. To ensure satisfactory joints in the metallized paper, *it is strongly recommended that simple "stuck" joints be not used*, but that the joints be held together firmly by wooden battens, as shown in Figure I. The method of screening the doors is shown in Figure II.

6. *Floor* (see Figure I).—The floor should be covered with wire gauze (½-in. or ¼-in. mesh galvanized iron wire), the screening material being in good electrical contact with the metal covering the walls. An earth connexion should be made to the floor screen by soldering thereto an earth wire of stranded copper, 7/·029 (7/22 S.W.G.), which should be connected to a cold-water pipe or buried earth-plate. The floor screen should then be covered with linoleum or other similar floor-covering material.

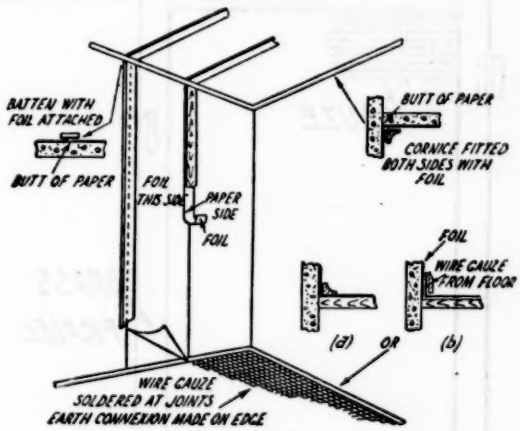

FIGURE I.
Method of screening walls, ceilings and floors.

7. *Windows.*—Figures III and IV show the only method practicable at present of screening a window; the window opening is covered with ¼-in. by ¼-in. wire gauze, supported on a metal frame making intimate contact with the screening material of the room. The use of glass reinforced with ¼-in. by ¼-in. wire gauze would be satisfactory. Provided the edges of the glass overlap the metal frame by an adequate amount.

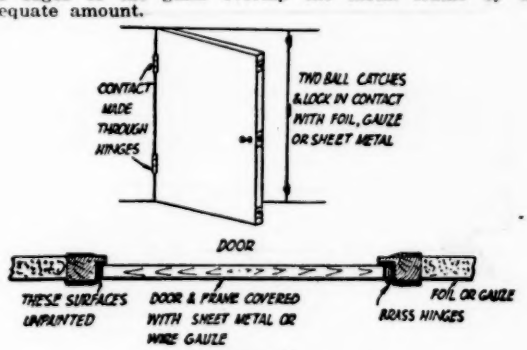
CONTACT MADE THROUGH HINGES
TWO BALL CATCHES & LOCK IN CONTACT WITH FOIL, GAUZE OR SHEET METAL
DOOR
THESE SURFACES UNPAINTED
DOOR & FRAME COVERED WITH SHEET METAL OR WIRE GAUZE
FOIL OR GAUZE
BRASS HINGES

FIGURE II.
Method of screening a door.

8. *Decorating.*—Many types of distemper and paint may be applied as a finish to the metallized paper surface. The information given in the following paragraphs has been supplied by the courtesy of a well-known firm of paint manufacturers:

> "The problem of painting or distempering aluminium foil would appear to fall under two heads, firstly, the question of obtaining satisfactory adhesion and, secondly, the possibility of chemical reaction between the aluminium foil and certain ingredients that may be contained in the paint or distemper.
>
> In the first case, the adhesion may be affected by the presence of traces of grease on the surface of the foil. In the manufacture of foil, it is general to use a small percentage of some such substance as tallow when polishing the surface. The presence of traces of this will ruin the adhesion of an oil paint, but will have little effect on a water paint. Fortunately, it is comparatively easy to get over this question by going over the wall with a rag dipped in spirit or solvent."

(Another large paint-maker expresses the opinion that it is important that all traces of paperhanger's paste be

removed from the surface before paint is applied, and that the aluminium foil should be perfectly dry before treatment.)

"The special primer, to give the necessary adhesion for oil paint, should then be applied.

Water paints are not affected so easily by the traces of grease on the surface to be painted, and we do not think it necessary to go over the walls with a solvent

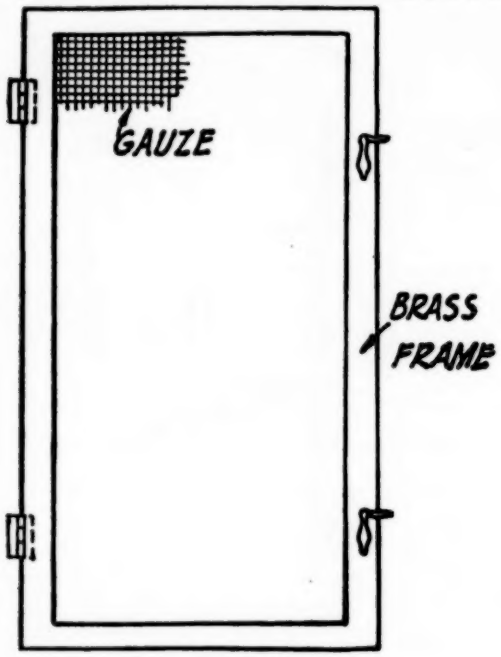

FIGURE III.
Method of screening a window.

when using this method of painting, but the second problem is likely to crop up with water paints, as many of them contain alkaline matter to which aluminium is very susceptible. For instance, soda ash, lime, borax &c., will react with aluminium."

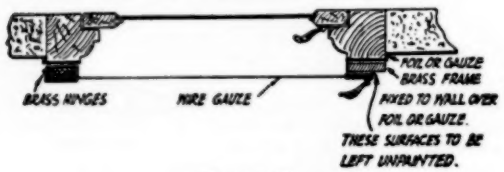

FIGURE IV.
Method of connecting the screening of a window to the screening of the room.

Screening a Room during its Construction.

9. Installation of the screening at this stage permits the use of wire gauze, which is preferable (from a life aspect) to paper-backed foil.

10. *The Stage at which to begin Screening.*—Screening work should commence at that stage in the building work when the fabric of the building is completed, but before the woodwork is completed, doors are fitted, walls and ceiling plastered, or floor—if of composition or cement—is laid.

11. *Walls, Ceiling and Floor.*—The wire gauze should be fixed to the walls, ceiling and floor before plastering. Joints should overlap about 1 inch, and a soldered connexion between adjacent strips of gauze should be made *every* 12 *inches.* An earth lead consisting of a 7/·029 stranded copper conductor (7/22 S.W.G.) should be run from a good earth such as a cold-water pipe, and soldered at two or three places to the wire gauze covering the floor.

12. *Door(s).*—The door(s) may be covered with metallized paper as shown in Figure II, or wire gauze may be sandwiched between two thin layers of wood, or the wooden door may be covered with sheet metal. Connexion at hinges, lock and ball catches should be made as shown in Figure II. Hinges and locks should be made of a non-rusting metal such as brass, and should not be painted. If desired, a metal door-frame may be employed to facilitate bonding.

13. *Windows.*—The method of window screening described in paragraph 7 is suitable. It is important that good connexion be made at frequent intervals between the window screen and the wire gauze covering the walls.

14. *Decorating.*—When the screen is completed, the walls and ceiling should be plastered. The plaster used should not have a strong "lime" or alkaline reaction, as this would corrode the wire gauze. The floor covering (i.e., composition, tiles or linoleum) should then be laid. The floor screen should be protected from damage by boards and sacking until the permanent covering material is laid. Any normal finish, such as distemper, paint or wall-paper, can then be applied to the walls and ceiling. Wire gauze screens fitted over windows should be treated with oil paint or plated.

Erecting a Screened Cubicle in a Large Room.

15. *General.*—If diathermy or therapy apparatus is to be used in one restricted portion of a large room, either existing or projected, the cheapest effective method of screening is to build a comparatively small screened cubicle within the large room. The method of screening will be similar, generally, to that described in paragraphs 4 to 14. The most inexpensive arrangement is to place the cubicle in a corner of the room and utilize portions of two walls of the room as walls of the cubicle after covering them with metallized paper. In this way only two cubicle walls will have to be built and screened.

Necessity for Mains Filters.

16. It is essential that all electric conductors entering the screened room or cubicle should be "filtered". A suitable filter, comprising chokes and condensers, is shown in Figure V. The filter should be fitted inside, as close as possible to the wall of the room, through which the supply leads are to be run. *All* supplies in the room, e.g., power supply to electro-medical apparatus, lighting and heating, must be taken from the filter. If differing tariffs apply to lighting and power, it will be necesary to employ two separate mains filters, one for lighting and the other for power. The object of screening may be defeated if, for example, an unfiltered pendant light is led through the ceiling screen.

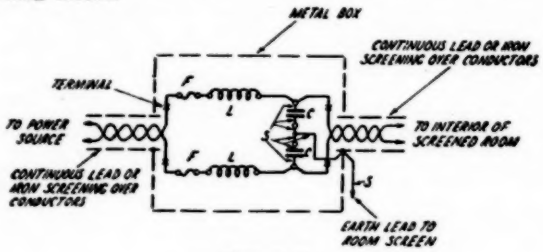

FIGURE V.
Screened filter unit.

Notes.

(*a*) L. 100 μH approx.; pie wound; low self capacity; air core.
C. 0·1 microfarad in parallel with 0·001 microfarad. Voltage rating chosen for continuous operation at the voltage of the power source.
F. Fuses: size governed by amount of power drawn by screened room equipment.
(*b*) Where maximum filtering is not required it may be possible to omit inductances L.
(*c*) Leads marked S to be as short as practicable.
(*d*) Locate filter preferably on one of the screened surfaces and at or near ground level.

17. The type of filter required, and consequently the cost, depends on the total power rating of the apparatus in the room. An example of a typical installation is given below:

Diathermy unit		300 watts
Lighting	say	200 "
Heating	"	2,000 "
Total		2,500 "

If the supply is obtained at 200 volts, the resultant current will be approx. $\frac{2,500}{200}$ = 12·5 amp. The rating of the filter required will therefore be 200 volts, 12·5 amp. It is customary to rate filters in volts and amperes—not watts.

18. It is appropriate to mention here that electrical heating of screened rooms is strongly advocated; other types of heating impair, and may even nullify, the effect of screening. Hot- or cold-water pipes and gas pipes leaving a screened room provide an escape path for the interference, which can then be radiated outside the room. Any piping which must enter the room should be well earthed and bonded to the screening material.

Gas and solid-fuel fires must be avoided, as the flues required for the products of combustion cannot be screened.

Installation of screened filter unit (Figure V) constitutes electrical wiring work and shall be the subject of notification to the supply authority.

APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 50, March 15, 1941.

CITIZEN NAVAL FORCES OF THE COMMONWEALTH.

Royal Australian Naval Reserve.

Appointment.—Jack Dennis Isles is appointed Surgeon Lieutenant, dated 15th February, 1941.

Promotion.—Surgeon Lieutenant James Aloysius Foedus Flynn is promoted to the rank of Surgeon Lieutenant-Commander, dated 1st March, 1941.

AUSTRALIAN IMPERIAL FORCE.

Australian Army Medical Corps.

To be Colonel.—Major W. A. Hailes, D.S.O., and to be Consulting Surgeon, Australian Imperial Force in the Middle East, 12th December, 1940.

To be Lieutenant-Colonel.—Lieutenant-Colonel D. C. Pigdon, E.D., and to command a Convalescent Depot, 1st January, 1941.

To be Majors.—Captain (Temporary Major) H. L. Andrews, 1st January, 1941; and Honorary Captain E. T. T. Downie, 1st February, 1941.

To be Captains.—Captain N. A. Walker and Honorary Captains H. H. B. Bradley and R. D. Puflett, 1st January, 1941.

To be Captains.—Captains V. G. Bristow, 1st February, 1941, F. N. Street, J. M. Oldham, R. H. Kaines and A. V. Day, and Honorary Captains R. K. Wilson, J. W. H. Merry, D. G. Alsop, J. W. Sangster, V. H. Vernon and V. E. Sampson, 1st January, 1941; William Patrick Ryan and Kenneth James Grice, 1st January, 1941, and Tom Charlton Anthony, 17th January, 1941.

To be Captain.—Honorary Captain W. R. Lane, 18th December, 1940.

Captains R. J. Humphery and J. C. Stewart are transferred to Reinforcements, 1st December, 1940, and 17th January, 1941, respectively.

The following officers are transferred from Reinforcements from the dates shown: Captains G. Read, 15th December, 1940; P. N. O'Donnell, 1st January, 1941; and V. H. Vernon, 18th January, 1941.

To be Major.—Captain R. F. K. West, 18th December, 1940.

AUSTRALIAN MILITARY FORCES.

NORTHERN COMMAND.

First Military District.

Australian Army Medical Corps.

Lieutenant-Colonel N. C. Talbot, M.C., E.D., relinquishes command of a Field Ambulance, 11th December, 1940.

To be Majors (temporarily).—Captains R. B. Charlton, J. A. McGree and J. W. Ralston, 13th November, 1940.

Australian Army Medical Corps Reserve.

To be Honorary Captains.—John Robert Adam, David Charles Fison, Clive William Kingston, Edward Joseph Ryan, 30th January, 1941.

EASTERN COMMAND.

Second Military District.

Australian Army Medical Corps.

To be Captain (provisionally).—Alvin Graeme Robson, 14th January, 1941.

Australian Army Medical Corps Reserve.

To be Honorary Captains.—Calleghan John McCarthy King, 20th January, 1941; Gordon Hastings Hair, 22nd January, 1941; Kenneth Boulton Shallard and Carlyle Graham Crawford, 23rd January, 1941.

SOUTHERN COMMAND.

Third Military District.

Australian Army Medical Corps.

Major M. Jacobs, E.D., is appointed to command a General Hospital and is granted the rank of Lieutenant-Colonel (temporarily), 5th December, 1940.

Honorary Captain F. J. Cahill is appointed from the Reserve of Officers (A.A.M.C.), and to be Captain (provisionally), 31st January, 1941.

Fourth Military District.

Australian Army Medical Corps.

Honorary Captain R. A. Isenstein is appointed from the Reserve of Officers (A.A.M.C.), and to be Captain (provisionally), 31st January, 1941.

WESTERN COMMAND.

Fifth Military District.

Australian Army Medical Corps.

The following officers are appointed from the Reserve of Officers (A.A.M.C.), and to be Captains (provisionally), 29th January, 1941: Honorary Captains W243395 M. J. Morris, W28587 J. B. Hogg, W8 T. Godlee, C. W. Anderson, W28615 J. M. Flynn, W233392 R. G. Linton, J. A. R. Mitchell, D. D. McCowan, A. B. Webster, T. C. Boyd, B. W. Nairn, R. H. F. Rockett, W76 T. B. Seed, C. Fortune, M. Kelly, N. C. Joel, A. D. Smith, R. S. W. Thomas, E. R. Beech, H. M. Hill, S. N. Michaels, H. C. Callagher, A. F. Walsh, N. G. Row, and I. O. Thorburn.

MICROSCOPES WANTED FOR THE AUSTRALIAN IMPERIAL FORCE.

THE Minister for the Army, the Right Honourable P. C. Spender, has made an appeal to medical practitioners and scientific workers throughout Australia to give or sell to the Army their spare microscopes. Standard type microscopes, both monocular and binocular, with oil immersion lenses, cannot be bought in Australia in the ordinary commercial way. Persons willing to dispose of their microscopes are asked to communicate without delay with the Director-General of Medical Services at Army Headquarters, Melbourne.

Correspondence.

VENEREAL DISEASE.

SIR: The figures published in your issue of March 1, leading article and page 280, relating to the increasing prevalence of venereal disease in New South Wales are disquieting.

During the Great War 400,000 men were put out of action for five to six weeks from venereal disease. Anyone who understands army administration will realize what this meant in medical, orderly and supply requirements, and the appalling wastage of man power. I have publicly stated that the defeat of the Fifth Army in 1918 was largely due to this cause. It is true that the 400,000 cases were drawn from all troops—British and Dominion—and not from the Fifth Army alone.

Soon after their return from the Dardanelles the incidence rate in the Australian Army for a short period was 25%. When a final outburst amongst all troops occurred in Egypt in 1918, Field-Marshal Lord Allenby, under advice, adopted three principles which brought the disease under control: (i) moral persuasion through the Chaplain-General and his staff; (ii) social services (*i.e.*, clubs, hostels and the like) conducted by the superb Y.M.C.A. service in all parts of his field and even in the front line; (iii) adoption of thorough prophylactic measures by the whole of the medical service.

As set out in a work of mine, "A Vision of the Possible", at Port Said the results of the medical efforts were most impressive.

Men, and especially young men, being what they are, there is no total escape from venereal disease except by

prophylaxis. The other methods were of service; but one dealt with some men who said they had indulged in this way most of their lives and they did not care what generals, padres, Y.M.C.A. or doctors said; they intended to continue.

In civil life I have often stated that the chief remedy is an alteration of our social outlook and early marriage. Censuses show that venereal disease in women begins at the age of fifteen and reaches its peak at nineteen years of age. With men it commences rather later and reaches its peak at the age of twenty-one, so that early action is essential to be of value. Unpleasant as it may be, there is no good shutting one's eyes to the nature of the animal we have to deal with. In the case of syphilis, prophylaxis is not difficult; but the prophylaxis of gonorrhœa really requires skilled attention, as we found at Port Said.

Lastly, though there is no proof, there is the possibility, as we suspected, of the enemy in times of war subsidizing infected women. It is altogether an ugly and difficult problem, dangerous in time of war, and deadly enough in civil life, though not so serious.

Anyone who will read Volume II of "The Official History of the War" will find the problem fully set out and temperately discussed. It is an illuminating document, and anyone who reads it will realize how difficult it is—indeed almost impossible—to control human nature even when the evil is gross.

It is no time for hesitation; it is better in every way to face the issue.

Yours, etc.,

JAMES W. BARRETT.

103-105, Collins Street,
Melbourne, C.1,
March 10, 1941.

SERRATUS ANTERIOR PARALYSIS.

SIR: I thank Dr. G. Keith Smith for his comments on my article on *serratus anterior* paralysis complicating shoulder dislocation. In the case recorded the line of treatment carried out was by a "clover-leaf" sling in a position of adduction, which is essentially the line of treatment Dr. Smith advocated.

The abduction splint was suggested as an alternative procedure because, while dislocated shoulder needs only three weeks' rest in adduction, the *serratus anterior* paralysis needs three to six months. If the arm were kept adducted for this time after dislocation, adhesions and contractions would cause permanent limitation of abduction. It is to avoid this complication that I ventured to suggest the abduction splint even though this is not ideal in treating dislocated shoulders. In this position, with flexion of the forearm, the "winging" of the scapula disappears, as was demonstrated by Horwitz and Trochantins, and rest secured for the paralysed *serratus anterior*.

The action of the *serratus anterior* in movement of the shoulder is very well described by A. Lee McGregor in "A Synopsis of Surgical Anatomy". He maintains that the *serratus anterior* is capable of rotating the scapula and elevating the arm from the horizontal to the vertical plane, the action of the trapezius being to provide a fixed point for the rotation. He also records a case demonstrated by Sir Harold Stiles, in which, in the presence of trapezius paralysis, the *serratus anterior* was able to perform the second 90° of abduction if a fulcrum was provided for the *serratus magnus* by pressing the scapula against the chest wall so that it could rotate but not move sidewards.

Yours, etc.,

GEORGE C. V. THOMPSON.

17, Collingwood Road,
Newport, W.15,
March 8, 1941.

SOME MEDICAL ASPECTS OF CRIME.

SIR: There are a few points in various criticisms of my article on "Some Medical Aspects of Crime" which call for a reply.

Dr. Guy Griffiths (THE MEDICAL JOURNAL OF AUSTRALIA, January 25, 1941) makes three brief critical suggestions which are quite justified and which I gratefully accept. Sir James Barrett (THE MEDICAL JOURNAL OF AUSTRALIA, February 8, 1941) appears to think that I wish to carry leniency to the criminal to a point where it becomes a menace to the community. If that can be fairly deduced from my article, then I expressed myself badly, my main theme being that the safety of the community is very ill served by the present system of punishment, and that it is worth while to inquire whether a more rational method, in the light of modern psychological knowledge, might not be evolved. The following quotations from my article show, I think, that I did not forget the need to protect society.

> Society, obviously, must use whatever amount of restraint is needed to protect itself from anti-social beings; but the point is that the present method, dominated by the idea of punishment, does not achieve it.

And, in discussing sexual criminals:

> If cure is possible they become useful citizens; if not, they must be restrained permanently.

"What could be plainer than that?" as Henry VIII is said to have remarked when he first saw Anne of Cleves.

Dr. S. F. McDonald, in the journal of March 22, 1941, asks: "Why does the critic of British institutions find it so necessary to drag in the marvels of Soviet Russia as a contrast?" And on March 8, 1941, Dr. Paul G. Dane heartily supports him. That Dr. McDonald's question is coupled with a claim to a scientific outlook surprises me, for surely the essence of a scientific outlook is to consider every available fact, to weigh all the evidence, and to use all the comparisons which are relevant. I take it that the question is intended to refer to institutions in general, and answer it in that sense.

If, in examining sociological conditions in all the countries with an advanced capitalist economy (that is, the countries which have entered the monopolist stage of capitalism), one sees the same picture, a picture of limitation of output, unemployment, under-consumption, and destruction of "surplus" products, it is surely reasonable and useful to try to see what is happening in the only country which has a socialist economy. A perfectly commonsense method should enable us to determine beyond reasonable doubt what evidence to believe and what to distrust. If one finds that the same general picture is given by such people as the Dean of Canterbury, Sidney and Beatrice Webb, Professor Laski and Dr. Sigerist, and if one then finds that these are people of unquestioned integrity, and in addition that they spent times varying from months to years in the U.S.S.R., and in addition to that that they were specially qualified to carry out their investigations, is not one justified in accepting their statements as, in general, correct? On the other hand, if we find a startlingly opposite picture drawn by, say, Trotsky, Souvarine and Krevitsky, are we not wise to remember that the first of these was exiled for treason, another expelled from the Communist Party, and the third of such obscure origin that it is impossible to say for certain who or what he was, except that if he was ever in Russia he was exiled, and to suspect that the picture may well be distorted by hatred, and the artists not altogether to be relied upon? It seems to me that there is no comparison in the credibility of the two groups.

My view, then, is that, where it is possible, in every sociological investigation the corresponding conditions in the U.S.S.R. should be also investigated for the sake of comparison. I did not do so fully in the article in question, partly because of length and partly because I realize that at the present time dispassionate consideration of such conditions is unfortunately impossible to many. I hardly know whether or not to regret the five lines of comparison which did slip in and appear to have caused such consternation.

Yours, etc.,

E. P. DARK.

Katoomba,
New South Wales,
March 11, 1941.

FLUOROGRAPHY.

SIR: In your March 1 issue is published an article on "Fluorography" by H. Gough. Just what does it all mean, and how does it add to the total of human knowledge?

He admits his costs are only guesses. Proved results of 35 millimetres may be read in many journals and books. In fairness I ask you to publish the following facts, confirmation of which in regard to the 35 millimetres can be easily obtained locally from military and air force authorities. It would be interesting to know where similar information can be obtained in regard to the 4 × 5.

1. The price of £1,100 is for the 4 × 5 tunnel, camera and screen only. The equivalent in 35 millimetres costs £300. The price mentioned, £900, for the 35 millimetres includes the X-ray generator and tunnel complete. Therefore, for £1,100, for a single 4 × 5, one could buy three (3) equivalent 35-millimetre tunnels, cameras and screens.

2. The operating, processing and recording costs of the 4 × 5 are no less than the ordinary 17 × 14 X ray. The 35 millimetres is 40% less in processing, 50% in operating, and about 80% in filing, recording and interpreting, according to methods used. The 35-millimetre material costs are one-sixth of the 4 × 5.

3. The generally recognized authority in the Commonwealth, whose services are available for the asking, is Dr. Eddy, Physicist-in-Charge of the Commonwealth X-Ray and Radium Laboratory. He has made fundamental researches into miniature X-ray work. His findings correspond to those of overseas physicists.

4. Highest medical opinion conveyed in current medical books and journals confirms that of physicists. Seth Hirsch, Professor of Radiology, University of New York, in *Radiology* of January 1, 1941, makes no mention of 4 × 5.

5. The 4 × 5 size has in itself no special merits. The 35-millimetre is standard movie size, and does not require any special hangers, cassettes and processing. The same detail seen at the picture theatres is available on the 35-millimetre X ray, under proper conditions. The re-raying of about 3%—not 10%—of the 35 millimetres on 17 × 14 is simply due to an army regulation. It is well known that of the 51,000 men sent from Victoria, not one has been returned on account of T.B.

6. There is only one manufacturer of 4 × 5, who is overseas. This firm, as well as their local representative, is now buying up all they can get, and advertising for 35-millimetre, 1·5 Leica and Zeiss cameras. The 35 millimetres, with the exception of the camera and screen, is completely made in Australia and has been proved by X raying all the forces in the Commonwealth. On the first day of mass radiography in Australia, over 2,000 men were X rayed and films processed at Ingleburn on January 1, 1940, by one 35-millimetre X-ray machine. Not one-tenth of this number could have been done using the 4 × 5, which entails the same processing methods and work, and costs, except materials, as the 17 × 14.

7. "However, fluorographic surveys on 35 m.m. films fulfil every diagnostic requirement, permitting the determination of the earliest lesions both in the lungs and lymph nodes. The routine and methods of its application are simple, practical and rapid, and the cost is small. The tempo and cost are important in mass surveys. With the introduction of this fluorography, which simplifies and materially reduces the cost of the examination, it becomes possible to think of the examination in universal terms, and thus the roentgenologist enters his third and most important phase in human betterment." (Hirsch, "Fluorography", page 11.)

Yours, etc.,

L. PARER.

British Medical Association House,
135, Macquarie Street,
Sydney.
March 11, 1941.

THE CRITERION OF CURE IN PURULENT MENINGITIS.

SIR: In your journal of March 1, 1941, at page 274, you discuss one form of purulent meningitis, and in the last paragraph stress the need to be certain that cure is complete before treatment is stopped. With this view I most emphatically agree, but you give us no reliable criterion which will guarantee for us that relapse will not occur. Even though "the patient is apyrexial and well, and a lumbar puncture has yielded fluid from which organisms cannot be cultured", still relapse sometimes occurs. The difficulty with purulent meningitis these days is not how to treat it, but to know how long to keep treating it. This question, of course, can only be answered by experience, and since meningitis is not a common disease, we will reach a conclusion more rapidly if we all pool our experience. May I suggest, therefore, that everyone who treats meningitis, and particularly the children's hospitals, should collect and publish data on this specific problem. At the time of cessation of sulphonamide therapy we should record information on at least the following points: (i) the general clinical condition of the patient, (ii) the period for which the temperature has been normal, (iii) the characters of the cerebro-spinal fluid, with particular reference to its cell, protein and glucose content and to the results of culture, with a note of the time and nature of previous findings.

The record, of course, must state the identity of the infecting organism, the severity of the infection and whether convalescence proceeded to recovery or whether relapse occurred. The problem is a separate one for each type of meningitis. Thus it is safe to prophesy that the criteria of cure of meningococcal meningitis will be very much less strict than of influenzal, in which the tendency to relapse is notorious. I think it likely that from the above points we will be able to establish a satisfactory standard of cure, but if any of your readers can suggest any other observations that are likely to be of value I will be glad to know them, for it is time we made a deliberate cooperative attempt to solve this difficulty.

Yours, etc.,

D. G. HAMILTON.

Royal Alexandra Hospital for Children,
Sydney
March 13, 1941.

THE FREEDOM OF THE MEDICAL PROFESSION.

SIR: To accept Dr. Brown's suggestions for a voluntary restriction of the freedom of the members of the medical profession is to accept a counsel of despair. Under the present theory of money and finance, which recognizes debt, usury, unemployment, malnutrition, bankruptcies, insolvencies, and recurrent depressions both great and small as inevitable, it is no doubt better for us to organize ourselves to fit into such conditions than to be organized by someone else. But is the existing theory of money and finance inevitable? It is surely as open to revision and reconsideration as any other of the works of man.

"By their fruits ye shall know them": and by its fruits our financial system is clearly worse than a barren tree—it bears poisonous fruits which set class against class and nation against nation and leads to nations running amok—to "Insanity Fair". Individuals who behave so destructively as whole nations now do are certified and immured for their protection and our own, but they are recognized to be ill. Nations surely are ill too who act as all the world is doing now! Why are they ill? Because they have never learned how to live satisfyingly together, and they have not learned to do this because their system of exchange (money and finance) is an exploiting system demanding 5% profit—rooted in war, flowering in war, profits take the place of spoils.

For a nation, and the nations, to live at peace and fruitfully with each other, exploitation must be abandoned and a fair exchange adopted, an exchange which gives a pound for a pound's worth, a pound's worth for a pound! Such a fair exchange can be secured by exchanging work for work; this can be done by using a money based on work-time, and recognizing every kind of work as eligible for credit. A new theory of money and of finance is necessary for the welfare of the world and for a successful end to the war, to an ending that will not breed new tyrannies worse perhaps than the old.

It is Australia's job to investigate this theory, and for her medical men to realize that the sacrifice of their freedom will not abolish poverty, malnutrition, and the tax of ill health on patients—such evils will only be confirmed and perhaps increased by the enslavement of medical men and women to the Moloch of finance; whereas an exchange of work for work (through a work-time money—Wice currency) will make all workers without exception vocational workers, while debt, usury, unemployment and the consequent malnutrition, become things of the past.

Guidance, and not "control", should be the aim of our organization. Control destroys freedom and with it personal initiative. Think of the Apple and Pear Board applied to medicine!

If we aim at a free vocational medicine we free others also; if we achieve "control" we enslave others as well as ourselves. It is not the machine that has enslaved us and our society, it is the money we use which gives the ownership of the machine to others than the users, which enslaves us. Let us demand an inquiry into the theory of money!

Yours, etc.,

MARY C. DE GARIS.

Geelong,
Victoria,
March 16, 1941.

Obituary.

INGLIS HALL COWLING.

WE regret to announce the death of Dr. Inglis Hall Cowling, which occurred on March 17, 1941, at Melbourne, Victoria.

Nominations and Elections.

THE undermentioned has applied for election as a member of the New South Wales Branch of the British Medical Association:

Murray, Alexander Francis, M.B., B.S., 1940 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

The undermentioned has applied for election as a member of the Western Australian Branch of the British Medical Association:

Minc, Solomon, M.D., 1930 (Univ. Rome), Walcott Street, Mount Lawley.

The undermentioned has applied for election as a member of the Tasmanian Branch of the British Medical Association:

Kyle, Oswald Theodore, M.B., B.S., 1938 (Univ. Melbourne), Geeveston.

Australian Medical Board Proceedings.

TASMANIA.

THE undermentioned have been registered, pursuant to the provisions of the *Medical Act*, 1918, of Tasmania, as duly qualified medical practitioners:

Edwards, Dudley, M.B., B.S., 1940 (Univ. Melbourne), Public Hospital, Launceston.

Moore, Francis Morrissey, M.B., B.S., 1940 (Univ. Melbourne), Royal Hobart Hospital, Hobart.

Currell, James Holmes, M.B., B.S., 1940 (Univ. Melbourne), Royal Hobart Hospital, Hobart.

Medical Appointments.

Dr. Geoffrey Campbell Middleton has been appointed Government Medical Officer at Culcairn, New South Wales.

* * *

Dr. John Dale has been appointed to be a member of the Dental Board of Victoria, in pursuance of the provisions of the *Medical Act*, 1928, of Victoria.

* * *

Dr. Arthur Murray Cudmore has been appointed President of the Medical Board of South Australia, under the provisions of the *Medical Practitioners Act*, 1919-1935, of South Australia.

* * *

Dr. Richard Alexander Denniston Pope, pursuant to the provisions of the *Navigation Act*, 1912-1935, has been appointed to act at Onslow, Western Australia, as Medical Inspector of Seamen.

* * *

Dr. William Bruce Kirkland has been appointed as Senior Medical Officer, Grade 2, Second Division, Medical Services, Health and Meat Inspection Division, Northern Territory (Darwin).

* * *

Dr. John Lawrence Gorman has been appointed Second Assistant Medical Superintendent, Brisbane Mental Hospital, Goodna, Queensland, in pursuance of the provisions of *The Public Service Acts*, 1922 *to* 1924, and *The Mental Hygiene Act of* 1938, of Queensland.

* * *

Dr. Beatrice Alice Warner has been appointed as Medical Officer, Ante-natal Clinics and Venereal Diseases, Department of Health and Home Affairs, in pursuance of the provisions of *The Public Service Acts*, 1922 *to* 1924, and *The Health Acts*, 1937 *to* 1939, of Queensland.

Books Received.

"Studies on the Human Thyroid in Iceland", by Júlíus Sigurjónsson; 1940. London: H. K. Lewis and Company Limited. Demy 8vo, pp. 130, with 10 illustrations. Price: 6s. net.

"Experimental Studies of Ephemeral Fever in Australian Cattle", by I. M. Mackerras, M.B., Ch.M., B.Sc., M. J. Mackerras, M.B., M.Sc., and F. M. Burnet, M.D., Ph.D.; 1940. Melbourne: Commonwealth of Australia, Council for Scientific and Industrial Research. Medium 8vo, pp. 116, with illustrations.

Diary for the Month.

APR. 1.—Queensland Branch, B.M.A.: Post-Graduate Committee.
APR. 1.—New South Wales Branch, B.M.A.: Council.
APR. 2.—Western Australian Branch, B.M.A.: Council.
APR. 2.—Victorian Branch, B.M.A.: Branch.
APR. 3.—South Australian Branch, B.M.A.: Council.
APR. 4.—Queensland Branch, B.M.A.: Branch.
APR. 4.—Victorian Branch, B.M.A.: Legislation Subcommittee.
APR. 8.—New South Wales Branch, B.M.A.: Organization and Science Committee.
APR. 8.—Tasmanian Branch, B.M.A.: Branch.
APR. 9.—Queensland Branch, B.M.A.: Brisbane Women's Hospital Clinical Society.
APR. 10.—Victorian Branch, B.M.A.: Ethics Committee.
APR. 15.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
APR. 15.—New South Wales Branch, B.M.A.: Ethics Committee.
APR. 16.—Western Australian Branch, B.M.A.: Branch.
APR. 17.—Queensland Branch, B.M.A.: Ipswich Hospital Clinical Society.
APR. 17.—New South Wales Branch, B.M.A.: Clinical.
APR. 17.—Victorian Branch, B.M.A.: Executive.
APR. 18.—Queensland Branch, B.M.A.: Council.
APR. 22.—New South Wales Branch, B.M.A.: Medical Politics Committee.
APR. 23.—Victorian Branch, B.M.A.: Council.
APR. 24.—Queensland Branch, B.M.A.: Brisbane Hospital Clinical Society.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135, Macquarie Street, Sydney): Australian Natives' Association; Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Proserpine District Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178, North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205, Saint George's Terrace, Perth): Wiluna Hospital; all Contract Practice appointments in Western Australia.

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